Congress Book – Symposia Proposals
42nd EWCBR – Les Diablerets

SYMPOSIUM 01 Sleep, motivation and fatigue

Chair(s): John Axelsson (john.axelsson@su.se)

Abstract: This session will cover the role of sleep and fatigue for motivation, behaviour, and our capacity to judge our own performance. 1) Tina Sundelin will present recent data on the role of sleep for metacognition (i.e. thinking about one's own thinking). Sleep usually leads to impaired performance in several cognitive domains, but to what extent is that mirrored in how aware people are of their own performance deficits? Can we trust our evaluation of our own abilities when sleep-deprived? 2) John Axelsson will present data and a model for how we assure recovery after exposure from different stressors, with focus on motivation and involved mechanisms. 3) Ida Nilsen will present data showing how sleep health dimensions associate and predict different phenotypes in motivation and arousal. This talk will cover the sleep health patterns that best predict groups characterized with e.g. anhedonia, high approach or calmness. 4) Martin Jonsjö will present data from an ongoing study on avoidance of internal aversive sensations in chronic fatigue conditions.

Speakers:

Title: Sleep and metacognition

Tina Sundelin, tina.sundelin@psychology.su.se, Stockholm University, Sweden

Abstract: The relationship between sleep and metacognition (i.e. thinking about one's own thinking) will be explored. Sleep usually leads to impaired performance in several cognitive domains, but to what extent is that mirrored in how aware people are of their own performance deficits? Can we trust our evaluation of our own abilities when sleep-deprived?

Title: Assuring recovery after different stressors: how sleepiness and inflammation alter motivation

John Axelsson, john.axelsson@su.se, Stockholm University, Sweden

Abstract: Sleep and fatigue are important homeostatic drives assuring recovery from a wide range of stressors. In this talk, I will present data and a model for how we assure recovery after exposure from different stressors, with focus on motivation and involved mechanisms.

Title: Mapping sleep – motivation relationships using cluster analyses

Ida Nilsen, ida.nilsen@su.se, Stockholm University, ,

Abstract: Motivation varies widely across individuals in both intensity (for example high or low arousal) and direction (for example engaging in social or physical activities). This talk will cover how sleep health dimensions associate and predict different phenotypes in motivation and arousal.

Title: Mindset interventions modulating bodily responses.

Mats Lekander, mats.lekander@ki.se, Department of Clinical Neuroscience, Karolinska Institutet, Stockholm, Sweden

Abstract: Mindset theory suggests that core beliefs (mindsets) about situations (e.g., stress or health interventions) can shape outcomes. For instance, having a "stress-is-enhancing" mindset (believing that stress can improve performance or health) is linked to better health, lower stress-hormone

reactivity, and improved performance under stress. Importantly, these mindsets are malleable: brief interventions (such as watching educational videos) have, in large-scale preregistered studies, successfully shifted stress mindsets, reduced anxiety, improved academic performance, and altered physiological responses. The present talk will discuss whether and how mindset interventions can be applied to the domain of inner bodily (inflammatory) stress. If so, predictive coding could provide a useful framework for understanding such effects. In the context of illness or vaccination, if a person expects to feel very sick or fearful, this top-down expectation could amplify the perception of symptoms and stress. Conversely, if one expects that "these symptoms are a positive sign that my body is working", the same physical stimuli might be appraised as less threatening and could influence attitudes such as willingness to vaccinate.

SYMPOSIUM 02 The Glio-Vascular Brain: A Dynamic Interface Driving Adaptation, Plasticity, and brain repair

Chair(s): Sylvie REMAUD (sremaud@mnhn.fr)

Abstract: Glial cells are not a passive "glue", but are recognized as the fundamental interface through which the brain integrates and responds to environmental, metabolic, vascular, and hormonal cues. These glial responses involved astrocytes, microglia, and oligodendrocyte to integrate signals from endothelial cells, smooth muscle cells, pericytes, perivascular macrophages, fibroblasts and neurons to shape brain plasticity, behavioral outcomes, and the brain's capacity for adaptation and repair.

This symposium aims to highlight an integrated view of glia as an adaptive system supporting brain function across contexts, stressors and multiple vertebrate species. Across diverse experimental models, from aquatic organisms to mammals, we will show how physiological stressors (hypoxia, metabolic and osmotic challenges, endocrine fluctuations), microbiota-derived metabolites and environmental pollutants, can functionally modulate neuroglial dynamics and their functional consequences. In particular, we will illustrate the central role of the glia cells in coordination with other brain cells in the behavioral outcomes, and how it can be modulated for drug development. Specifically, Dr. Boris Zalc will illustrate how the evolutionary history of myelin and its remarkable repair capacity can be leveraged through amphibian models, showing how external environmental cues, notably the forever chemicals (PFAS), affect oligodendrocyte dynamics and myelin integrity Complementing this perspective, lifelong endocrine influences, particularly thyroid hormones, emerge as powerful regulators of glial fate, sculpting oligodendrocyte differentiation and myelin formation and maintenance across development and aging (Dr. Sylvie Remaud). Extending beyond intrinsic physiology, Dr Jérôme Badaut will show how neurovascular unit plasticity offers a mechanistic lens to understand how environmental constraints, including exposure to PFAS can alter vertebrate behavior through glial–vascular remodeling. Finally, Dr Hirt will demonstrate how microbiota-derived metabolites, including SCFAs and urolithin A, modulate stroke outcome through effects on microglial activation, astrocytic responses, and blood–brain barrier integrity.

Together, these presentations illustrate the diverse and dynamic pathways through which glia and brain vessels integrate environmental, hormonal, and metabolic signals to influence brain function and health across the lifespan.

1)Dr Boris Zalc (ICM, Paris): From myelin origins to repair mechanisms: lessons from amphibian models
2)Dr Sylvie Remaud (MNHN, Paris): Lifelong endocrine regulation of glial cells: how thyroid hormones shape oligodendrocyte fate
3)Dr Jerome Badaut (CEBC, CNRS, Villiers en Bois): Applying Neurovascular unit plasticity to explain behavioral traits in vertebrates under environmental constraints: example of new generation of eternal pollutant
4)Dr Lorenz Hirt (UNIL-CHUV, Lausanne): Pomegranates, whole grain oat porridge and stroke outcome

Speakers:

Title: From myelin origins to repair mechanisms: lessons from amphibian models,

Zalc Boris, boris.zalc@icm-institute.org , ICM Paris,

Abstract: Myelin is synthetized by Schwann cell in the peripheral nervous system (PNS) and by oligodendrocyte in the central nervous system (CNS). We have proposed that during evolution myelin was acquired by Placoderms the earliest branch of the jawed fishes 425MY ago. In fishes myelin composition is very similar in the PNS and CNS using P0 (myelin protein zero; mpz) as the most abundant protein. Mammals use P0 as a major PNS myelin protein and in contrast P0 is absent from CNS myelin being replaced by PLP and MBP. This major differences between PNS and CNS myelin proteins occurred in amphibian. To investigate drugs that could favor remyelination or on the contrary, substances that may interfere with myelination physiology, we have developed a transgenic Xenopus Tg(mbp:gfp-ntr) of conditional demyelination. In this transgenic line the mbp regulatory sequence drive very selectively the expression of the GFP-NTR transgene into myelin forming oligodendrocyte. Thanks to the GFP reporter mature oligodendrocytes fluoresce in green. Ntr encodes the E. Coli nitroreductase, which convert molecules such as metronidazole harboring a NO2 radical into a highly cytotoxic hydroxylamine derivative. As a result, adding metronidazole into the swimming water of tadpole induces mature oligodendrocyte cell death and a severe demyelination. Spontaneous remyelination occurs upon returning the tadpole into normal water, well advanced after 3 days and nearly complete after 8 days. Thanks to the transparency of Xenopus tadpoles these events (demyelination and remyelination) can be monitor in the optic nerve of live animals using a fluorescent macroscope. At the end of demyelination, addition into the swimming water of candidate drugs favoring remyelination accelerate the recovery of GFP positive oligodendrocyte in the optic nerve. We have successfully used this transgenic line to screen for candidate drugs favoring remyelination and have identified pro-remyelination molecules such as siponimod, retinoic acid or clemastine. Using the same strategy we have been able to demonstrate the deleterious effect of some PFAS interfering with remyelination. In addition we reasoned that demyelination should translate into loss of sensori-motor functions followed by behavioural recovery upon remyelination. To this end we measured the swimming speed and distance travelled before and after demyelination and during the ongoing spontaneous remyelination and have developed a functional assay based on the visual avoidance of a virtual collision.

Title: Lifelong endocrine regulation of glial cells: how thyroid hormones shape oligodendrocyte fate

Remaud Sylvie, sremaud@mnhn.fr, CNRS/MNHN UMR7221

Abstract: Thyroid hormones (THs) are central regulators of neural stem cell (NSC) fate within the subventricular zone (SVZ), orchestrating the balance between neuronal and glial lineages from development through ageing. Converging evidence shows that intracellular control of TH availability—

through TH transporters, deiodinases, and TH binding proteins—defines the temporal windows during which oligodendrocyte progenitor cells (OPCs) acquire lineage identity, and initiate myelination. Our work spanning the perinatal period, young adulthood, and ageing demonstrates that THs serve as stage-specific gatekeepers: they shape early SVZ fate decisions, regulate the neuron–glia switch through coordinated transcriptional and translational programs, and later sustain oligodendrocyte maturation and long-term behavioural outcomes. Disruption of TH signaling—via genetic perturbations, pharmacological inhibition, or endocrine-disrupting chemicals — changes OPC specification, impairs differentiation, compromises myelin formation, and disrupts regenerative capacity.

Recent studies further reveal that TH signaling integrates metabolic cues, local niche factors, and injury-induced pathways, positioning THs as lifelong determinants of oligodendrocyte plasticity. Together, these findings outline an endocrine framework in which finely tuned TH dynamics govern the emergence, maintenance, and resilience of myelin-forming cells, with implications for neurodevelopmental vulnerability, adult myelin homeostasis, and susceptibility to demyelinating disease.

Title: Applying Neurovascular unit plasticity to explain behavioral traits in vertebrates under environmental constraints: example of new generation of eternal pollutant

Badaut Jérôme, jerome.badaut@cebc.cnrs.fr CNRS UMR 7372, Centre d'Études Biologiques de Chizé

Abstract: Wild vertebrates face diverse environmental constraints that shape their behavior and ultimately influence fitness. Yet, the brain mechanisms supporting such adaptive responses are poorly understood, understudied and often reduced to neuronal alterations while overlooking other brain cells. The neurovascular unit (NVU) is composed of endothelial cells, astrocytes, pericytes, and neurons and represents a dynamic interface that regulates exchanges between the brain and the periphery. The two last decades, It is increasingly recognized in laboratory models as a key modulator of behavior. However, its potential role in the adaptive responses of wildlife to environmental stressors remains unexplored. We therefore propose that NVU plasticity is a central mechanism allowing vertebrates to adjust their behavior to ecological challenges. To illustrate this concept across multiple species exposed to distinct natural constraints, we tested the effects of salinity fluctuations in green frogs (Pelophylax sp.), the repeated chronic hypoxia and sleep restriction in southern elephant seals (Mirounga leonina), and the pollutant exposure in yellow-legged gulls (Larus michahellis) and lab mouse (mus musculus).

The NVU plasticity has been tested in various models of vertebrates: 1) In wild green frogs inhabiting ponds of varying salinity, we detected presence of GFAP in plasma for the first time in a wildlife context. GFAP levels were higher in frogs from more saline ponds, suggesting that astroglial/NVU responses to osmotic stress are reflected in peripheral biomarkers. Moreover, captivity induced a sex-specific GFAP modulation, highlighting the sensitivity of NVU-related signals to environmental variation. In a second animal model, southern elephant seals (SES) exposed to chronic diving-related hypoxia, adults exhibited pronounced NVU structural remodeling compared to post-weaning pups, including ~62% higher vascular volume in grey matter, increased vessel branching and tortuosity, and disruptions in large vessels. These features resemble vascular phenotypes described in human aging and neurodegenerative conditions, suggesting an early neurovascular fragility under repeated hypoxic stress, associated with increase of the neurofilament light chain (NFL) in the plasma of SES coming from the ocean trip. In a third case study of emerging contaminants, we assessed the neurobiological effects of the PFAS-degradation product 7:3 fluorotelomer carboxylic acid (7:3 FTCA). In yellow-legged gull embryos, environmentally relevant exposure induced pallium-specific increases in IBA1 and GFAP, decreased AQP4 signaling, and reduced cytokine expression, revealing selective NVU vulnerability. This result was confirmed in C57Bl/6J mice fed with 7:3 FTCA, associated with elevated neuronal calcium activity in the prefrontal cortex and increased open-arm exploration in the elevated plus maze, indicating altered prefrontal circuit function. These cross-species findings show that emergent PFAS disrupt NVU-related homeostasis and neural activity.

Together, these studies demonstrate that diverse constraints—chemical pollution, hypoxia, and osmotic stress—consistently induce remodeling and plasticity of the NVU across vertebrates. This supports the emerging view that the NVU is a central, plastic interface through which environmental factors shape brain function and behavioral traits, and underscores the urgent need to evaluate novel pollutants and ecological stressors through this neurobiological lens.

Title: Pomegranates, whole grain oat porridge and stroke outcome

Lorenz Hirt, Lorenz.Hirt@chuv.ch , Neurology, Department of clinical neuroscience, CHUV and Department of Fundamental Neuroscience, Lausanne University

Abstract: The gut microbiome is now recognized as a significant modulator of disease in various conditions including stroke. Recently, metabolites produced by gut bacteria, termed gut metabolites, were shown to influence the outcome after stroke. Our lab is examining the role of some specific gut metabolites in brain injury, neuroinflammation and barrier integrity in a model of ischemic stroke in the mouse.

Short chain fatty acids (SCFAs) result from the fermentation by certain gut bacteria of alimentary fibres contained in whole grain cereals or legumes. I will present results from our lab on the impact of SCFA supplementation on stroke outcome and the puzzling phenomenon of gut bacteria translocation and dissemination after stroke in mice. Indeed, in the brain hemispheres, bacterial material was detected by immunofluorescence only within the ischemic lesion, in vicinity of activated microglia, surrounded by a rim of activated astrocytes. This phenomenon was attenuated by SCFA treatment.

SYMPOSIUM 03 From brain development to addiction: social and neural circuit mechanisms, plasticity, and therapeutic targets

Chair(s): Emmanuel Darcq Co Chair: Victor Mathis

Abstract: Early-life alcohol exposure profoundly reshapes brain development, neural circuits, and behavior, contributing to neurodevelopmental disorders and addiction. Sophie Laguesse will present cross-species evidence identifying NR2F1 as a conserved molecular mediator of cortical migration defects and sensory dysfunction induced by prenatal alcohol exposure. Victor Mathis will discuss how social context and isolation reshape neural processing of social information and modulate vulnerability to opioid and psychostimulant use. Yvan Vachez will describe alcohol-induced synaptic adaptations within ventral pallidal subthalamic circuits that may drive compulsive drinking and inform deep brain stimulation strategies. Finally, Emmanuel

Darcq will highlight the habenula as a key hub linking opioid signaling, negative affect, and relapse, and will discuss novel orphan GPCR-based therapeutic approach. Together, these talks bridge development, circuits, and behavior to identify actionable targets for addiction and neurodevelopmental disorders.

Speakers:

Title: NR2F1 is a Cross-Species Mediator of Cerebral Cortex Defects Induced by Fetal Alcohol Intoxication

Sophie Laguesse, slaguesse@uliege.be
Institution: Université de Liège, Belgium Authors: S. Laguesse, Charlet-Briart M, Van Hees L, Oskera L, Boutsen A, Bonafina A, Lavergne A, Reyskens C, Close R, Epifanova E, Helgueta S, Didone V, Tielens S, Studer M, Nguyen L.

Abstract: Prenatal alcohol exposure (PAE) remains the leading preventable cause of neurodevelopmental disorders, contributing to a spectrum of cognitive, sensory, and behavioral deficits collectively known as Fetal Alcohol Spectrum Disorders (FASD). To date, the precise mechanisms by which PAE disrupts brain development remain unclear, limiting the development of targeted therapies and effective prevention strategies. Alcohol is particularly harmful to the cerebral cortex, and individuals with FASD frequently exhibit impaired sensorimotor function, including deficits in tactile perception.

Using a mouse model of voluntary binge-like alcohol consumption during gestation, we demonstrate that PAE disrupts the migration and connectivity of callosal projection neurons in the developing somatosensory cortex and induces long-lasting sensory deficits. We identified NR2F1, a key transcription factor involved in neuronal migration and cortical patterning, as a novel alcohol-sensitive target mediating these effects. Mechanistically, PAE leads to upregulation of NR2F1, together with downregulation of its direct targets Lis1 and Kif1b, which are essential for neuronal migration and differentiation. Importantly, these molecular alterations are conserved in the developing cortex of human fetuses with documented prenatal alcohol exposure, as well as in human cerebral organoids engrafted into ethanol-exposed mice.

Together, our findings reveal a conserved molecular and cellular pathway disrupted by PAE, highlight NR2F1 as a key regulator in the pathogenesis of FASD, and suggest new avenues for therapeutic intervention targeting alcohol-induced neurodevelopmental impairments.

Title: Social context matters: Neural processing of social information in substance use disorders

Victor Mathis, victor.mathis@unistra.fr
Institution: INSERM, U1329 STEP, Strasbourg, FRANCE

Abstract: Despite the widespread use of social media and digital communication platforms, loneliness and social isolation have become increasingly prevalent in modern societies. In the context of substance use disorders, the social environment of individuals plays a crucial role and can, by itself, influence patterns of drug consumption, drug effects and their consequences, and even contribute to relapse. Indeed, one of the strongest predictors of substance use disorders is the presence of drug users within one's social circle, including family members and friends. Furthermore, the deterioration of the social environment among individuals with substance use disorders is well documented, with many reporting profound feelings of social isolation and loneliness.

However, how a central nervous system altered by drug exposure processes and integrates social information remains poorly understood. In addition, the potential facilitating effects of social isolation on substance use and relapse vulnerability require further investigation. In this talk, recent findings on the effects of opioid use (morphine) on social behaviors will be presented, as well as original data demonstrating that social isolation directly modulates central responses to psychostimulant drugs (cocaine) and associated drug-taking behaviors in murine models.

Title: Subthalamic circuit adaptations in alcohol use disorder: implication for deep brain stimulation?

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Keywords: Alcohol addiction, Subthalamic Nucleus, Ventral Pallidum, Synaptic Plasticity, Deep Brain Stimulation.

Abstract: Alcohol addiction is a chronic relapsing disorders characterized by the compulsive use of alcohol.

Despite decades of research, there is no efficient treatment available for alcohol addiction. In this context, deep brain stimulation (DBS) is a hot topic of great interest in the field. Subthalamic nucleus deep brain stimulation (STN-DBS) has shown promising results, consistent with the behavioral inhibition role of the STN. However, the optimal stimulation parameters remain to be determined in order to selectively modulate the pathological behaviors, minimizing side effects. Characterizing the STN circuits altered by alcohol intake, particularly those mediating the compulsive use will ultimately facilitate the development of more targeted DBS protocols.

We first aimed to characterize the projections from the Ventral Pallidum (VP) to ventromedial territory of the STN (vmSTN). The VP is mainly inhibitory (GABAergic), crucial for reward processing, and its activity is altered following ethanol consumption. We hypothesized that enhanced inhibitory transmission between the VP and the STN may promote compulsive use. Using a mouse model of ethanol drinking, we combined in vivo electrophysiology, patch clamp recordings, pharmacology and neuromodulation including chemogenetic and optogenetic, to examine ethanol-induced alterations in the VP to vmSTN synaptic strength. Our findings provide new insights in the STN circuitry, and will pave the way for behavioral investigations of VP to vmSTN activity modulation, along the characterization of the STN-DBS effects onto that circuit.

Title: Habenular control of mood in naive and opioid dependent animals: new therapetic strategies

Emmanuel Darcq, edarcq@unistra.fr
Institution: INSERM, U1329 STEP, Strasbourg, FRANCE

Abstract :Opioid use disorder represents a major public health challenge, with relapse and negative emotional states as key drivers of chronicity. The mu opioid receptor (MOR) plays a central role in balancing pleasure and distress, yet how MOR-expressing neurons regulate these emotional states remains unclear. The habenula, a key brain region linking reward and aversion, emerges as a critical node in this process. Using genetically identified MOR neurons in the habenula (HbMOR), we investigated how their activity shapes emotional responses.

We combined fiber photometry, opto- and chemogenetic approaches in Oprm1-Cre knockin mice to monitor and manipulate HbMOR neuron activity. We first measured their responses during blockade of endogenous and exogenous opioid signaling with naloxone. We then tested whether inhibiting these neurons could alleviate aversive states during conditioned place aversion in both naïve and morphine-dependent mice. Finally, we pharmacologically manipulated oGPCRs enriched in the habenula to reduces opioid withdrawal.

Naloxone administration increased HbMOR neuronal activity, after high doses in naïve mice, and even at low doses in opioid-dependent mice, suggesting enhanced habenular sensitivity during dependence. Importantly, chemogenetic inhibition of HbMOR neurons reduced aversive behavior in both contexts.

These findings identify opioid-sensitive habenular neurons as key modulators of negative emotional states. By linking

opioid signaling to aversion, this work highlights the oGPCR enricehd into the habenula as a potential target for alleviating emotional distress associated with opioid withdrawal and other mood disorders.

SYMPOSIUM 04 Role of expectations in shaping symptoms, behavior, and clinical outcomes

Chair(s): Julie Lasselin (julie.lasselin@ki.se)

Abstract: Expectations fundamentally shape how symptoms are perceived, how individuals respond to bodily signals, and how they recover, in both clinical and everyday contexts. This symposium brings together experimental, ecological, mechanistic, and clinical perspectives to demonstrate the pervasive role of expectations in health. The talks will show how expectations, whether learned, violated, maladaptive, or deliberately targeted in treatment, profoundly influence symptoms, behavior, and clinical outcomes.

Speakers:

Title: Shaping of sickness expectations via social learning,
Julie Lasselin, julie.lasselin@ki.se , Karolinska Institutet / Stockholm University / Linköping University, Sweden,
Abstract: Sickness expectations might play a critical role in symptom experience and recovery, yet little is known about how they can be shaped through social learning. This two-part study aimed to identify and validate video stimuli capable of manipulating sickness expectations in the context of an experimental endotoxemia study. In the first phase, focus groups were used to qualitatively identify behavioral and contextual cues that signal different levels of sickness and coping. In the second phase, we are experimentally testing the impact of these videos on expectation ratings in a convenience sample of colleagues. Participants view randomized triads of videos and report how sick they expect to feel in a similar situation. We will present findings on how observed symptom expression influences participants' own sickness expectations, with the goal of establishing reliable video-based manipulations for future psychoneuro-immunological studies.

Title: Prediction errors and human behavior in the wild: insights from naturalistic data,
Leonie Balter, leonie.balter@su.se ,Stockholm University / Karolinska Institutet, Sweden,
Abstract: We know from laboratory studies that prediction errors, the difference between what was expected and what was experienced, play a role in learning and decision making. But does this principle scale to real-world behavior, where outcomes are noisy, irregular, and far less controlled? To address this question, we analyzed naturalistic data from anglers on fishing trips and tested whether prediction errors forecast their subsequent decisions. This provides insight into how prediction-error learning mechanisms play out in real-life behavior.

Title: Negative expectations, fear, and hypervigilance in visceral pain: Mechanisms and clinical implications in functional and chronic-inflammatory bowel diseases,
Sigrid Elsenbruch, sigrid.elsenbruch@rub.de ,Ruhr-Universität Bochum, Germany,
Abstract: Chronic visceral pain poses a major challenge for patients with functional and chronic-inflammatory bowel diseases. Fear, hypervigilance, and negative expectations toward gastrointestinal symptoms are transdiagnostic mechanisms that amplify pain perception, trigger nocebo effects, and reduce quality of life. This talk will explore the underlying neurobiological and psychological mechanisms, using fear conditioning as a translational research model to illustrate how negative cognitions, fear and hypervigilance interact with central and peripheral pain pathways along the gut-brain axis. Clinical implications will be discussed, including evidence-based psychological treatment strategies, with a focus on visceral pain-specific techniques within cognitive-behavioral therapy approaches that target maladaptive expectations and symptom-related anxiety.

Title: Expectation mapping in cognitive behavior therapy
Kristoffer Månsson kristoffer.mansson@ki.se, Karolinska Institutet, Sweden
Abstract: Patient expectations are central to therapeutic processes and outcomes, yet their measurement remains limited in scope in psychiatric treatments. In this study, we introduce a novel approach which quantifies treatment expectations at the level of specific symptoms. Prior research has typically assessed expectations globally, overlooking the nuanced and heterogeneous nature of anticipated change across symptom domains. We applied this approach in the context of a clinical trial of internet-delivered cognitive behavior therapy (CBT) for individuals with social anxiety disorder (SAD). Before treatment initiation, participants reported their expectations regarding improvement for distinct symptoms. Preliminary results will be presented on inter-individual variability in symptom-specific expectations, with some symptoms perceived as more amenable to change than others. We will further examine how these expectation profiles relate to clinical outcomes. Our initial results support the clinical utility of expectation mapping as a tool for both research and personalized treatment planning. This method may enhance our understanding of expectancy effects and may facilitate precision in CBT delivery.

SYMPOSIUM 05 Psychopathology and Stress as Dynamic Processes: From Neural Variability to Multilevel Regulation

Chair(s): Nicolas Rohleder (nicolas.rohleder@fau.de)

Abstract: Psychopathology and stress-related phenomena are traditionally studied using static markers, such as mean neural activation, single biological indicators, or cross-sectional symptom measures. However, both experimental and clinical evidence increasingly suggest that stress responses and psychiatric symptoms emerge from dynamic processes that unfold over time and across interacting brain–body systems. Capturing these dynamics is essential for advancing mechanistic understanding and for bridging neuroscience with biological psychology.
This symposium brings together four complementary contributions that conceptualize stress and psychopathology as dynamic, multilevel processes linking neural, biological, behavioral, and computational levels of analysis. One contribution adopts a cognitive neuroscience perspective to examine the progression of psychosis, framing symptom development from prodromal to chronic stages within a predictive coding account of altered information processing. Extending this process-based view, another contribution demonstrates that moment-to-moment neural variability during socioemotional processing robustly differentiates individuals with social anxiety disorder from healthy controls and generalizes across independent samples, outperforming conventional mean activation measures. At the biological level, evidence is presented showing how early-life adversity in Borderline Personality Disorder shapes cellular energy metabolism, highlighting dynamic regulation rather than fixed vulnerability. Finally, full-body movement dynamics

during acute psychosocial stress are shown to predict stress exposure and hypothalamic–pituitary–adrenal axis reactivity using machine-learning approaches, illustrating how behavioral dynamics can serve as scalable, non-invasive markers of stress processes.
Together, the contributions illustrate a shared conceptual shift away from static snapshots toward dynamic, multilevel accounts of stress and psychopathology. By integrating perspectives across levels of analysis, the symposium highlights the value of process-based approaches for understanding brain–body interactions in health and disease.
Speakers:

Title: Toward generalizability of moment-to-moment neural variability as a diagnostic biomarker of social anxiety disorder

Kristoffer N T Månsson, kristoffer.mansson@ki.se, Karolinska Institutet, Sweden

Abstract: Background: Robust neural biomarkers are essential for precision psychiatry, yet most fail to generalize out-of-sample, limiting clinical utility. Moment-to-moment neural variability, reflecting dynamic fluctuations, has emerged as an alternative to mean activation measures, but its diagnostic potential remains largely unexplored.
Methods: We tested whether socioemotional neural variability differentiates social anxiety disorder patients from controls and generalizes to independent samples. Functional magnetic resonance imaging was acquired during a socioemotional face- and object-matching task from a discovery sample (80 patients, 48 controls) and a validation sample (26 patients, 25 controls). Partial least squares modeling was applied to whole-brain voxel-wise variability and activation maps. Generalizability was assessed by applying stable discovery sample weights to the validation sample's independent data.
Results: Socioemotional neural variability significantly differed between groups in the discovery sample (permuted $P < .001$, Cohen's $d = 1.64$) and this generalized to the validation sample (permuted $P = .038$, $d = 0.59$). Neural variability also differed between faces and objects (permuted $P = .001$, $d = 0.59$) and showed in-sample associations with task accuracy (permuted $P = .037$, Pearson $r = 0.53$) and symptom severity (permuted $P = .005$, $r = 0.60$). Conventional mean BOLD response did not yield significant or generalizable group differences. All key neural variability effects remained robust across reduced data volumes, supporting feasibility for brief clinical protocols.
Conclusions: Socioemotionally induced neural variability may serve as a generalizable biomarker of social anxiety disorder. These findings support neural variability as a promising candidate for precision psychiatry and encourage research on its utility for treatment monitoring.

Title: From Symptoms to Cells: Immunocellular Bioenergetics in Borderline Personality Disorder

Felix Neuner, felix.neuner@uni-ulm.de

Title: Progression of psychosis from a cognitive neuroscience perspective

Predrag Petrovic, predrag.petrovic@ki.se, Karolinska Institutet, Sweden

Abstract: Psychotic disorders, such as schizophrenia, impose a substantial clinical and societal burden. Despite this, the neurocognitive mechanisms that give rise to hallmark psychotic symptoms—most notably delusions and hallucinations—remain incompletely understood. Extensive research has examined the genetic, cellular, and molecular bases of psychosis; however, because psychotic symptoms ultimately manifest as alterations in behaviour emerging from disrupted information processing, it is equally essential to investigate these phenomena at the computational and cognitive levels.
Cognitive neuropsychiatry seeks to explain abnormal behaviour by identifying aberrations in underlying cognitive processes, thereby offering insights into why specific symptoms arise. Individuals in the prodromal stage of psychosis frequently progress to an early psychotic state characterised predominantly by positive symptoms, including hallucinations and delusions. Over time, the condition often evolves into a more chronic form marked by negative symptoms, such as social withdrawal and cognitive impairments. Understanding these developmental trajectories is therefore critical for elucidating the mechanisms underlying psychotic disorders, for predicting clinical progression and for precision medicine interventions.
In the present project, we aim to investigate the progression of psychosis from a high-risk state—such as the prodromal phase—to early psychosis and ultimately to chronic psychosis, each stage defined by distinct clinical features. This work will be conducted within a predictive coding framework and will integrate analyses of both behavioural manifestations and their underlying neural mechanisms.

Title: Fast and Slow Gene Expression Changes in Blood Following Acute Social Stress

Hampus Grönvall, hamgro@miun.se, Mid Sweden University, Sweden

Abstract: Social stress is a risk factor for psychiatric disorders and also influences immune function. While it is known that acute social stress impacts the number of immune cells in circulation, the temporal dynamics of stress induced immune-related transcriptional changes in human blood remain unclear. To investigate changes in gene expression, we exposed 26 adults to the Trier Social Stress Test (TSST), and collected blood at baseline, as well as 5, 30, 60 and 90 min after stress. Whole-blood gene expression was profiled using a 5' targeted RNA-sequencing method (STRT). Differential expression was analyzed using linear and cubic models. We observed a total of 54 differentially expressed genes following stress. Fast responses, with a transient peak immediately following stress, were enriched for cytotoxic T cell, NK cell and dendritic cell functions (e.g., GZMB, GNLY, CCL4 and GZMA) and paralleled lymphocyte count changes. In contrast, gradual, linear responses without any evident peak were enriched for neutrophil related genes (e.g., FPR2, PLAUR, CXCR2, AQP9, and QPCT) and did not mirror neutrophil counts, indicating cell intrinsic transcriptional changes. From pathway and transcription factor enrichment analysss, IL-12 family mediated signaling is inferred as a central mechanism linking stress to immune gene regulation. Our results show that acute psychosocial stress induces both fast and slower changes in gene expression in different immune cell populations. The involvement of the IL-12-STAT4 axis and genes such as PLAUR and FPR2 suggests molecular mechanisms through which stress-related immune activation may contribute to vulnerability for anxiety and depressive disorders.

Title: Body movements as biomarkers: Machine Learning-based prediction of acute psychosocial stress and HPA axis reactivity

Nicolas Rohleder, nicolas.rohleder@fau.de, Friedrich-Alexander University Erlangen-Nürnberg (FAU), Germany

Abstract: Investigating acute stress responses is crucial for understanding the underlying mechanisms of stress and for developing objective assessment tools. Current approaches largely rely on self-reports, which are prone to bias, or on biological markers that often require invasive and resource-intensive laboratory procedures. Body posture and movement represent a promising, yet still underexplored, additional modality for stress assessment, as they are systematically altered under threat and negative emotional states.
In the present work, we examined whether full-body movement patterns during acute psychosocial stress can be

used to predict both subjective stress exposure and endocrine stress responses. Across two studies (Pilot Study: N = 20; Main Study: N = 39), participants underwent the Trier Social Stress Test (TSST) and a stress-free control condition (friendly-TSST; f-TSST) in randomized order while wearing inertial measurement unit (IMU)-based motion capture suits. In a subsample (N = 41), salivary cortisol responses were additionally assessed to index hypothalamic–pituitary–adrenal (HPA) axis reactivity.

Acute stress induction was associated with a reproducible movement pattern, characterized by reduced overall motion and longer periods of immobility. Machine-learning models trained solely on movement features were able to distinguish stress from control conditions with accuracies of 75.0 ± 17.7 % in the pilot study and 73.4 ± 7.7 % in the main study. Importantly, movement dynamics also predicted endocrine stress responses: classification of cortisol responders versus non-responders reached an accuracy of 65.2 %, and regression models predicted cortisol increases with a mean absolute error of 2.94 nmol/l.

Together, these findings demonstrate that body posture and movement contain meaningful information about both acute psychosocial stress exposure and HPA axis reactivity. Movement-based markers may therefore serve as objective, non-invasive proxies of stress responses and represent a valuable extension to established psychological and biological stress measures. This work provides the first systematic evidence that full-body movement analysis can link behavioral and endocrine components of the human stress response.

SYMPOSIUM 06 Social psychoneuroimmunology

Chair(s): Estherina Trachtenberg (et581@cam.ac.uk) and Lina Hansson (lina.hansson.2@ki.se)

Abstract: Social processes shape disease avoidance, immune regulation and health across development. This symposium brings together new work demonstrating how subtle facial cues of illness influence real-time social distancing, how individuals decide when to conceal sickness symptoms depending on social context and group identity, and how three-year-old children differ from adults in their capacity to mount proactive mucosal antibody responses to contagion cues. It also highlights data-driven approaches to mapping social heterogeneity in neurodevelopment, showing how prosociality relates to learning, communication and brain morphology, and presents an updated assessment of open science practices within psychoneuroimmunology, including challenges for transparency in sensitive research. Together, these presentations span behavioural, developmental, computational and methodological perspectives, providing an integrated view of how social perception, interaction and communication shape immune function and health. These contributions deepen our understanding of the mechanisms linking social environments and immunity, and outline key directions for strengthening theory, methodological rigour and translational relevance in social psychoneuro-immunology.

Speakers:

Title: Prosociality and social heterogeneity in neurodevelopment: a machine learning approach in CALM

Estherina Trachtenberg, et581@cam.ac.uk
Institution: MRC Cognition and Brain Sciences Unit, University of Cambridge, Cambridge, UK

Abstract: Social behaviour is an important but underexamined dimension of neurodevelopment. Children referred for attention, learning and memory difficulties often present with complex social challenges that do not align with traditional diagnostic categories, which motivates data-driven approaches capable of capturing heterogeneity at scale.

We analysed behavioural data from the Centre for Attention, Learning and Memory (CALM), together with a non-referred comparison group, using standardised social and communication measures (SDQ, CCC-2, Conners). To characterise multidimensional social functioning, we applied machine learning methods, including self-organising maps (SOM), to generate a topological representation of social profiles without imposing diagnostic constraints.

Prosociality emerged as a major axis of variation, showing strong associations with reduced learning problems ($r = -0.84$), fewer communication difficulties ($r = -0.56$) and lower overall diagnostic burden. Communication difficulties formed a densely correlated cluster ($r = 0.70–0.85$), and peer problems co-occurred with behavioural and communication challenges.

The SOM identified four robust social profiles, including prosocial, making-friends difficulty, rejection or bullying and general social difficulty. These profiles showed significant differences in cognitive performance, including working memory ($F = 4.43$, $p = 0.001$), phonology ($F = 2.99$, $p = 0.018$) and academic ability ($F = 2.63$, $p = 0.033$). Generalised additive models ($R^2 = 0.11$), showing that prosociality declines with age and is higher in girls and non-referred children.

To link behavioural heterogeneity with neurobiology, partial least squares (PLS) analysis of T1-weighted MRI identified a distributed pattern of brain morphology that covaried with prosocial behaviour, indicating convergence between data-driven behavioural profiles and structural variation.

Together, these results position prosociality as a central and potentially protective construct in neurodevelopment and demonstrate the value of machine learning approaches for mapping social heterogeneity.

Title: Sick faces trigger social avoidance in humans

Elahe Tavakoli-Berg. Email: elahe.tavakoli@su.se
Institution: Stockholm University/Karolinska Institutet, Sweden

Abstract: Humans have evolved adaptive strategies to reduce infection risk in social environments, yet it remains unclear whether subtle, ecologically valid facial cues of sickness elicit measurable avoidance behavior. This study tested whether experimentally induced sickness cues influence interpersonal distance preferences. Using facial photographs collected two hours after participants in an endotoxemia study received either endotoxin (sick) or placebo (healthy), 247 observers (mean age = 23.72; 134 women) completed a computerized interpersonal distance task. They adjusted the apparent proximity of each face to simulate preferred conversational distance and then rated each face's health. Observers also completed questionnaires assessing personal health, immune status, and perceived vulnerability to disease. Preliminary analyses indicate that participants keep greater distance from sick faces compared to healthy ones ($\beta = 0.519$, $p = 0.011$), and distancing increases as perceived sickness severity rises ($\beta = -0.029$, $p < 0.0001$). Individual characteristics of observers and the inflammatory responses of the photographed individuals showed limited associations with avoidance behavior. We will present evidence that subtle facial indicators of illness can shape real-time proximity decisions, supporting the role of proactive behavioral immune responses in social interaction.

Title: To conceal or not to conceal? – Investigating how risk of disease transmission and the outgroup status affect concealment of infectious disease in humans

Lina Hansson Email: lina.hansson.2@ki.se
Institution: Karolinska Institutet/Stockholm University, Sweden.

Abstract: Findings from the animal kingdom reveal that expressions of sickness may be flexible. For instance, male birds suppress sickness symptoms in the presence of a female. A previous study indicates that humans also conceal sickness symptoms from others to a large extent. Yet, much remains unknown about how social contexts shape such behaviors, and the current study thus aims to investigate if the social interaction type (in-person vs. virtual) and the social target's outgroup status affect the likelihood of sickness concealment. Participants (N= 1118) were recruited from Prolific and conducted a computerized task, during which they imagined that they had the flu. In the task, participants were presented with several social scenarios, including a photo of the person they would meet, either in-person or online, and stated how likely they would be to participate and conceal their sickness symptoms in each situation. The presented faces varied in ingroup/outgroup status. The results indicate that participants were more willing to participate (β = 20.2, $p<0.001$) and conceal their sickness symptoms (β = 2.8, $p<0.001$) in a virtual context than in an in-person context. Outgroup status of the social target did not affect sickness concealment behaviors. These findings suggest that sickness concealment behaviors are not only motivated by disease transmission risks.

Title: Proactive immune-activation in early childhood. Do three-year-old children show an increase in salivary antibodies when watching someone sneeze?

Jenny Sachtler, jenny.sachtler@uni-hamburg.de
Institution: Department of Biology, Neuroendocrinology Unit, Faculty of Mathematics, Informatics and
Natural Sciences, Institute for Animal Cell and Systems Biology, University of Hamburg, Germany

Abstract: Humans own a repertoire of several mechanisms to face the constant threat of pathogen transmission within human social groups. This comprises both physiological immune defense, such as antibodies released by mucosal tissue, and behavioral mechanisms that help to avoid a pathogen contact in the first place by detecting sickness cues (e.g., sneezing and coughing) triggering avoidance behavior and feelings of disgust. However, if unavoidable contagious situations occur (e.g., when being sneezed at someone nearby) a proactive immune response in the respiratory tract can be observed, i.e. increased release of secretory immunoglobin A (sIgA), which occurs before the actual pathogen contact. Nonetheless, current findings are limited to healthy adults (≥18 years), precluding any inference on a similar mechanisms in young children. Especially, children below the age of 4 years lack knowledge about contagious illness transmission and also show immature disgust, which fully develops by the age of 7. This raises the question of whether they are able to (1.) mount a proactive immune response to contagion threats, and (2.) whether this depends on the development of conceptual knowledge about disease transmission and the ability to verbalize feelings of disgust. In the current study, we tested a group of three-year-old (n = 23; mean age = 42.7 months; 9f/14m) and their caretakers (n = 23, mean age = 37.5 years; 20f/3m). The children and their caretakers watched a Relaxation Video with nature sequences followed by a Disease Video containing typical sickness cues (e.g., sneezing people). They provided a saliva sample immediately after each video using a children swab, resulting in a Baseline and a Disease video sample. Additionally, caretakers filled out various questionnaires of trait and state disgust, perceived disease vulnerability and also rate their aversion towards the Disease video. The children were interviewed about their understanding of the presented sickness cues, their perceived contagion risk and disgust feelings. The results show, that children still lack the ability to mount a proactive immune response to potential contagion threat, and also show an overall deficit in sIga secretion compared to adults. Further, three-year-olds, who described the Disease video as disgusting, also exhibited a more developed ability to correctly describe and imititate the video content. Additionally, these children also had caretakers who indicated more state disgust in relation to the Disease video. Against expectations, we found a decrease in sIgA secretion in adults after watching the Disease video, which also correlated with higher perceived disease vulnerability. Notable, caretakers with younger children showed the strongest decrease in sIgA, while also exhibiting the highest perceived disease vulnerability, suggesting that caretakers of younger children have a higher perception of being exposed to contagion threats, supposedly associated with their children, while being concurrently forced to care for their offspring, which may also explain the higher sIgA at baseline. While the study mainly focused on the mucosal antibody response in three-year-old children and thereby suggests that conceptual knowledge about illness transmission and disgustingness may be mandatory to mount a proper sIgA response to a contagion threat, our findings also propose a possible modulation in the antibody response of parents with younger children that opposes previous findings in nulliparous adults.

SYMPOSIUM 07 Cognitive Modulation of Physiological Responses

Chair(s): John Axelsson (john.axelsson@su.se)

Abstract: Physiological responses to illness, stress, and environmental threat are not solely determined by bottom-up biological processes but are powerfully shaped by cognitive factors such as expectations, learning, appraisal, and meaning. This symposium brings together four complementary contributions that examine how cognition modulates physiological responses across systems, species, and levels of analysis.

The first talk presents Balance-ACT, a psychophysiological intervention for post-COVID condition that integrates Acceptance and Commitment Therapy with principles of homeostasis, alongside objective assessments of muscle endurance. The second contribution introduces an animal model of sickness-related associative learning, demonstrating how conditioned expectations can elicit anticipatory physiological responses to treatment-associated cues. The third talk focuses on mindset interventions, showing how malleable beliefs about stress and bodily reactions can alter subjective experience, physiological reactivity, and health-relevant attitudes, potentially through predictive-coding mechanisms. The fourth contribution provides neuroimaging and immunological evidence that visual cues of respiratory contagion engage brain regions such as the anterior insula and amygdala, linking cognitive evaluation of threat to mucosal immune responses.

Together, these talks illustrate converging pathways through which cognition shapes physiological regulation, from learned expectations and belief systems to neural and immune mechanisms. The symposium highlights implications for understanding symptoms, improving interventions, and bridging basic and translational research in psychophysiology and health.

Title: Associative learning in an animal model of sickness behavior

Harald Engler harald.engler@uk-essen.de , Institute of Medical Psychology and Behavioral Immunobiology, Center for Translational and Behavioral Neurosciences, University Hospital Essen, University of Duisburg-Essen, Essen, Germany

Abstract: Psychological and physical symptoms of sickness are common side effects of immunotherapy and chemotherapy. Due to the unpleasantness of these symptoms, many patients develop negative treatment expectations during the course of therapy and often exhibit anticipatory physiological responses when re-exposed to treatment-associated contextual cues such as smell, taste, or visual stimuli. Despite their broad clinical implications, the molecular and neurobiological mechanisms underlying negative treatment expectations and their physiological consequences remain largely unknown. However, it is difficult to obtain such mechanistic insights in humans for ethical reasons. Therefore, we developed an animal model of sickness-induced negative treatment expectations in rats. The model is based on an associative learning paradigm combining the injection of bacterial endotoxin (lipopolysaccharide) as a sickness-inducing treatment (unconditioned stimulus, US) with the presentation of an unfamiliar taste as a treatment-associated cue (conditioned stimulus, CS). This talk will provide an overview of the characteristics of the model and offer first mechanistic insights into how associative learning shapes anticipatory physiological responses to sickness-related cues.

Title: Balance-ACT for post-COVID condition

Prof Trudie Chalder, Institute of Psychiatry, Psychology and Neuroscience, Kings College London Email: trudie.chalder@kcl.ac.uk

Abstract: Persistent symptoms such as fatigue, brain fog and breathlessness are common in post COVID condition (PCC), requiring innovative approaches to management and assessment. We developed Balance ACT, a psycho physiological intervention integrating Acceptance and Commitment Therapy with homeostasis principles, refined through qualitative research, a scoping review, and patient and public involvement (PPI). Patients (n = 19) and healthcare professionals (n = 12) endorsed its acceptability, and feedback informed the creation of manuals, mindfulness recordings, training materials and a fidelity measure. In parallel, we examined methods for assessing muscle endurance, comparing volitional and non-volitional techniques in healthy adults (n = 14). Volitional handgrip and quadriceps endurance showed good reliability (ICC = 0.76–0.81), while non volitional quadriceps endurance demonstrated excellent reliability (ICC = 0.92). Together, these strands demonstrate the value of combining psycho physiological interventions with robust, objective measures of muscle function. Balance ACT offers a promising holistic approach for PCS management, while non volitional endurance testing provides reliable assessment tools for populations unable to perform voluntary manoeuvres. Our current research will evaluate the clinical efficacy and cost effectiveness of Balance ACT. In addition, the mechanistic relationships between subjective and objective measures of fatigue, breathlessness and outcomes will be investigated.

Title: ACHOO: Respiratory contagion cues modulate brain & mucosal immunity

Esther Diekhof, esther.diekhof@uni-hamburg.de,
Department of Biology, Neuroendocrinology
Unit, Faculty of Mathematics, Informatics and Natural Sciences, Institute for Animal Cell and
Systems Biology, University of Hamburg, Germany

Abstract: This talk will present neuroimaging and immunological evidence showing how the brain and the mucosal immune system respond to visual cues of respiratory contagion. Sixty-two participants viewed short videos of contagious versus non-contagious everyday situations, while brain activity and salivary secretory immunoglobin A (sIgA) were monitored. Situations involving contagion cues – such as sneezing or visibly sick individuals – elicited stronger activation in the anterior insula and other regions of the neuroimmune
axis compared to scenarios displaying healthy humans. Additionally, increased insula activity tracked both perceived contagiousness and disgust and also scaled with increased sIgA release in saliva, supposedly linking the central evaluation of the contagion threat with the peripheral immune response. In contrast, the amygdala responded to the presence of any humans, regardless of sickness signs, suggesting a general alertness to potential contagiousness, and the amygdala activation also specifically scaled with disgust. Together, these findings outline a proactive neuroimmune pathway that may help humans to manage contagion risks. While the insula may integrate contagion cues to mobilize cognitive and physiological defenses, the amygdala appears to act as a general alerting system for social situations with a potential transmission risk.

SYMPOSIUM 08 Beyond humans: evolutionary neuroscience of communication

Chair: Mélina Cordeau, melina_cordeau@fas.harvard.edu , Harvard (USA) and ELTE (Hungary) university.

Abstract: Human language is often considered a unique cognitive faculty, defined by symbolic meaning, syntax, and combinatorial structure. Yet growing evidence from comparative neuroscience and animal communication challenges this view, suggesting that the building blocks of language may have deep evolutionary roots. This symposium brings together complementary perspectives from vocal communication, comparative neuroanatomy, and network neuroscience to explore how communication systems emerge, reorganize, and specialize across species under evolutionary and environmental pressures. We will integrate evidence from wild chimpanzees, non-human primates, and domestic dogs to address fundamental questions about the origins of syntax, the neural architecture supporting communication, and the impact of selection, both natural and human-driven, on communication networks. By combining behavioral experiments, vocal analyses, diffusion MRI tractography, and emerging structure-function approaches using awake fMRI, this symposium highlights how comparative models can bridge the gap between animal communication and human language. Together, these talks offer a network-based evolutionary framework for understanding how complex communication systems arise across species.

Speakers:

Title: Why study dogs to understand human language?

Mélina Cordeau, melina_cordeau@fas.harvard.edu, Harvard (USA) and ELTE (Hungary) university.Human language is often regarded as a uniquely human faculty, yet its evolutionary origins remain deeply unresolved. Domestic dogs provide a powerful and complementary comparative model to address this paradox. Shaped by thousands of years of domestication, dogs have been selectively tuned to attend to, interpret, and respond to human communicative signals, offering a rare opportunity to study communication-driven brain adaptations in the absence of language itself.

In this talk, I will present diffusion MRI evidence from a large-scale analysis of structural brain networks in 110 dogs across 16 breeds, revealing a robust and distributed network architecture relevant to communication. I will then show how this network organization differs between pre-modern and modern dogs, as well as between service dogs and released service dogs, highlighting the impact of human-driven selection and experience on brain connectivity. Finally, I will outline ongoing work integrating structural connectivity with awake functional MRI to link network organization to the processing of human vocal signals, providing a comparative framework for investigating the neural foundations of language.

Title: Call combinations and compositional processing in wild chimpanzees.

Simon William Townsend,
simonwilliam.townsend@iea.uzh.ch , University of Zürich (Switzerland).

Through combining meaning-bearing words into larger phrases, human language expresses an open-ended number of messages. This syntactic ability has been argued to be a key feature distinguishing language from other animal communication systems. However, recent observational and experimental evidence of syntactic-like structuring in monkeys has challenged this assumption and suggests syntax might rather be evolutionary more ancient. Comparable data in great apes, our closest-living relatives, are critical to validating this claim and reconstructing the more recent evolutionary history of syntax. I will review recent progress we have made addressing this issue in the vocal communication system of wild chimpanzees in Uganda. Firstly, through leveraging methods previously developed in the field of computational lingusitics to identify non-random word combinations (collocation analysis) and applying them to vocal data we established a repertoire of chimpanzee call combinations. Secondly, using playback experiments, we have also probed the meaning of a candidate combination identified from collocation analyses: the alarm-huu-waa-bark. Chimpanzees produce “alarm-huus” when surprised and “waa-barks” when recruiting other group-members during aggression or hunting. Existing behavioural data further suggest chimpanzees combine these two calls together, specifically when encountering an unexpected threat that requires recruitment such as when exposed to a snake. To confirm these findings and verify the meaning-bearing nature of this precise call combination, we played back an artificial call combination and both calls produced independently to wild-living chimpanzees in the Budongo Forest, Uganda. Chimpanzees (N=6) reacted strongest to the combination (alarm-huu +waa bark), showing longer responses, compared with both individual calls (alarm-huu or waa bark). These experimental results suggest the “alarm-huu + waa-bark” combination represents a compositional syntactic-like structure, where the meaning of the sequence is derived from the meaning of its comprising parts. Our work supports previous work in monkeys and indicates the cognitive building blocks underliying syntax were already present in our last common ancestor with chimpanzees and are perhaps even older

Title: Temporal lobe connectivity and the evolution of semantic cognition.

Katherine Bryant, katherinelbryant@gmail.com, CRPN Marseille (France).

The faculty of human language is possible due to long-distance connectivity in prefrontal and temporal cortex. The human temporal lobe, in particular, has undergone important remodeling since our divergence from our primate ancestors that is critical to our language faculties. However, the temporal lobe emerged early in primate evolution and may have played an important role in primate ecology. In this talk, I will discuss how the primate temporal lobe has been modified to lay the foundation for human semantic cognition, with a special focus on the role of tractography for elucidating these evolutionary specializations.

SYMPOSIUM 09 Cutting New Tracks: Fresh Pathways Through Neuroinflammation, Cognition, and Recovery

Chair(s): Neil HARRISON (harrisonn4@cardiff.ac.uk)

Abstract: This symposium brings together emerging research on the biological and psychological mechanisms linking inflammation, cognition, and persistent symptoms across clinical conditions. Harrison introduces novel diffusion-weighted MRI methods (SANDI) capable of detecting microstructural glial changes during experimentally induced systemic inflammation, offering a promising biomarker for neuroinflammatory processes. Hansson presents evidence that seasonal allergies are associated with increased depressive symptoms and elevated Th2-related cytokines during pollen exposure, highlighting inflammation-related mood vulnerability in allergic individuals. Van der Schaaf discusses theoretical and neurobiological models of mental fatigue—including roles for neuroinflammation and mitochondrial dysfunction—and outlines advances in neurocognitive assessment using ecologically valid, momentary measures. Collectively, these talks showcase interdisciplinary progress toward understanding and treating inflammation-related cognitive, emotional, and fatigue symptoms.

Title: Detecting Neuroinflammation in the Human Brain Using SANDI: Evidence from an LPS model

Prof Neil Harrison. Cardiff University, UK.
Email: harrisonn4@cardiff.ac.uk

Abstract: Systemic inflammation is increasingly recognised as a key contributor in psychiatric and neurodegenerative disorders, with glial cells playing a key role in transmitting immune signals to the brain. Inflammatory activation induces characteristic glial morphological changes, including soma enlargement, which may be detectable using diffusion-weighted MRI (DW-MRI). Building on our prior diffusion-spectroscopy findings showing inflammation-related increases in choline diffusivity, this study tested whether Soma and Neurite Density Imaging (SANDI), an advanced multicompartment DW-MRI model, can capture microstructural alterations induced by low-dose lipopolysaccharide (LPS). Eighteen healthy adults completed a randomised, crossover LPS–placebo study. Participants received intravenous LPS (1 ng/kg) or saline on separate visits, with MRI acquired 4 hours post-injection on a 3T Siemens Connectom system using a high-b DW-MRI protocol optimised for SANDI modelling. Regions of interest were defined in the insular cortex given its role in immune–brain communication and the anterior operculum. SANDI-derived soma radius (R_soma) and soma density (f_soma) were compared across conditions. LPS administration resulted in a significant increase in R_soma within the insula ($p = 0.001$; $d = 0.93$), with no corresponding effects in the control region or on soma density. Notably, insular R_soma correlated positively with white cell count, linking MRI-derived microstructural changes to peripheral inflammatory response. These findings provide preliminary evidence that SANDI is sensitive to glial morphological changes during acute systemic inflammation, supporting its potential as a non-invasive neuroinflammation biomarker.

Title: The effect of seasonal allergy on symptoms of anxiety and depression

Dr Lina Hansson, Karolinska Institutet/Stockholm University, Sweden
Email: lina.hansson.2@ki.se

Abstract: Seasonal allergy has been proposed as a risk factor for comorbid psychological disorders.
Inflammation-induced mood cha nges triggered by pollen exposure represent a potential mechanism. In this study, patients with seasonal allergies (N=42) and healthy controls (N=42) were assessed during and outside of the pollen season. Anxiety and depressive symptoms were measured with the Hospital Anxiety and Depression scale. Blood samples were analyzed for inflammatory markers (IL-4, IL-5, IL-6, IL-8, IL-10, IL-13, TNF-α, and IFN-γ). Patients with seasonal allergies displayed more depressive symptoms (B=1.57, $p<0.05$) and higher IL-5 concentrations (B=0.9, $p<0.05$) compared to controls during the pollen season, with no between-group differences outside the pollen season and no seasonal variation in controls. Patients also had higher IL-13 concentrations than controls across both seasons (B=0.52, $p<0.05$). The increase in IL-5 concentrations did not mediate the increase in depressive symptoms (B=0.23, p=.34). No differences were found for anxiety symptoms or remaining inflammatory markers.

Title: Neuroinflammatory and autoimmune mechanisms in fibromyalgia

Prof Eva Kosek, Karolinska Institutet, Sweden
Email: eva.kosek@ki.se

Abstract: Neuroinflammation is indicated in fibromyalgia (FM) based on findings of elevated levels of proinflammatory cytokines in the cerebrospinal fluid and positron emission tomography (PET) studies demonstrating upregulated cerebral translocator protein binding interpreted as (micro)glia activation. Recent evidence indicates autoimmune mechanisms in a substantial proportion of fibromyalgia subjects (FMS). Data from translational studies reveal that transfer of IgG from FMS but not from healthy controls (HC), induce a FM-like state in mice. The IgG from FMS bind to and activate satellite glia cells (anti-SGC IgG) in the dorsal root ganglia (DRG) and sensitize peripheral small and medium size neurons. We and others have shown that high levels of anti-SGC IgG in FMS are associated with higher pain intensity. Furthermore, a cluster of immune system-related proteins, with central roles in humoral immune response was upregulated in the blood of FMS compared to HC. In addition, higher levels of immune-related proteins such as CD79b, a protein necessary for Bcell receptor function, and CD4, a co receptor needed for T cell activation also involved in B-cell activation was found in FMS with high compared to low levels of anti-SGC IgG. The recent findings indicating neuroimmune and autoimmune mechanisms in FMS will hopefully lead to better diagnostic criteria identifying FMS potentially eligable to immunomodulatory treatments. The new insights may also open up for development of disease modifying treatments for FM, currently a pain condition without efficient treatment options.

Title: Fatigue and cognitive symptoms: Using new theoretical and biological models to improve neurocognitive assessments.

Prof Marieke van der Schaaf, Tilberg University, Netherlands.
Email: M.E.vanderSchaaf@tilburguniversity.edu

Abstract: Mental fatigue or the experience of "brain fog" after mental exertion is prevalent after various medical diseases and post-infectious diseases. Although these symptoms can impact daily and occupational functioning considerably, they are not always fully captured by traditional neurocognitive assessments. This highlights a need to rethink how we assess cognitive symptoms in fatigue conditions. In this talk, I will review several theoretical models of mental fatigue along with potential neurobiological mechanisms, including neuroinflammation and altered mitochondrial functioning, that may underlie cognitive symptoms after disease. This challenges the way we think about mental fatigue and how its assessment can be improved. Finally, I will discuss some new cognitive tasks for ecological momentary assessments that can be used to test the hypotheses that derive from these models.

SYMPOSIUM 10 Why dry january is critical: brain mechanisms and alcohol addiction

Chair(s): Christelle Baunez (christelle.baunez@univ-amu.fr)

Abstract: The symposium will cover our last findings related to alcohol use disorders in both rodents and human with behavioural approaches and fMRI

Speakers: Mickael Naassila, Univ Jules Verne Picardie, Amiens, France ; Christelle Baunez, Institut de Neurosciences de la Timone, CNRS & AIx-Marseille Université, Marseille, France ; Philippe de Timary, Univ Louvain, Bruxelles, Belgium ; Didier Grandjean, Université de Genève

Role of microbiota in alcohol use disorders
Philippe de Timary, Univ Louvain, Bruxelles, Belgium

Role of STN in addiction
Christelle Baunez

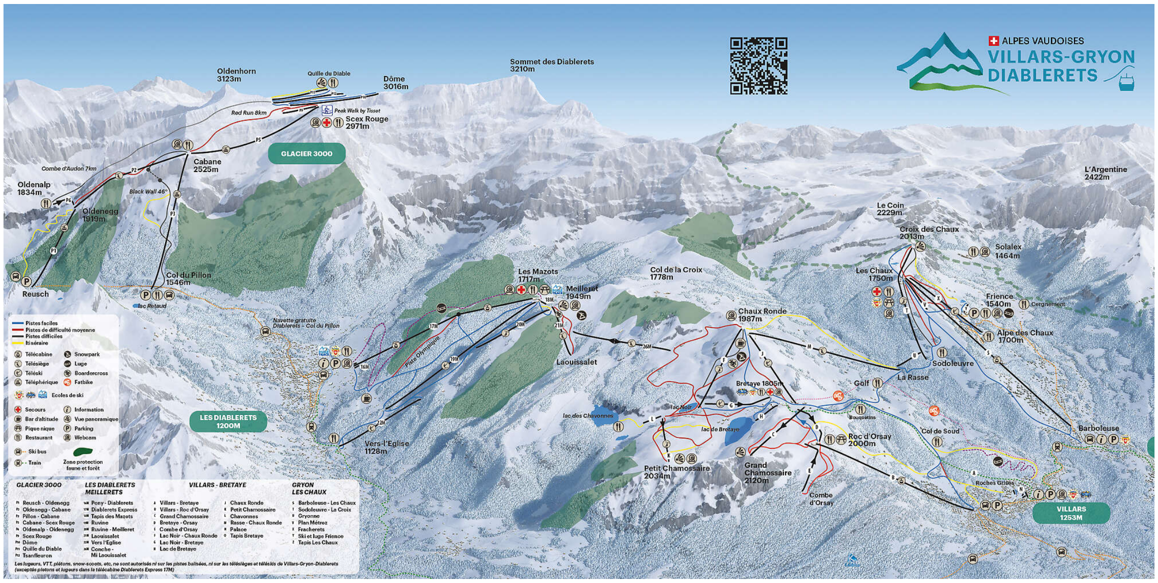
Title: Histamine H3 Receptor as a target for alcohol use disorder: challenging the predictability of animal models for clinical translation in drug development. Authors: Bernard Le Foll#, Mickael Naassila#, Jérôme Jeanblanc#, Christian S. Hendershot, Jesus Chavarria, Thierry Calmels, Stéphane Krief, Isabelle Berrebi-Bertrand, Marilyne Uguen, David Perrin, Xavier Ligneau, Isabelle Boileau, Pablo Rusjan, Patricia Di Ciano, Pamela Sabioni, Marc Capet, Philippe Robert Olivier Finance, Jeanne-Marie Lecomte, Jean Charles Schwartz.

Mickael Naassila, Univ Jules Verne Picardie, GRAP-INSERM UMR1247, Amiens, France.

There is an important need to advance medication development for alcohol use disorder (AUD). BP1.3656B, a highly potent and selective Histamine H3 receptor (H3R) inverse agonist/antagonist, has been developed. Preclinical studies revealed high affinity, good pharmacokinetic profile, good brain penetration, and favorable safety. BP1.3656B reduced alcohol drinking and alcohol-seeking behavior in rodents. Phase I studies revealed good tolerability/pharmacokinetic in humans. Positron emission tomography revealed high brain occupancy in humans. Based on this favorable profile, two trials were conducted in subjects with AUD. In non-treatment seekers, BP1.3656B had no impact on intravenous alcohol self-administration. A randomized clinical trial testing three doses of BP1.3656B versus placebo in treatment-seekers with AUD showed no reduction of heavy drinking days. Collective results illustrate the challenges inherent to clinical translation of AUD therapies, and reinforce the use of Phase IIa human laboratory paradigms as an important tool to de-risk translation of innovative drug targets for AUD.

Title: Social context and alcohol cues dynamically modulate inhibitory control in risky drinking via cortical and subcortical networks: evidence from fMRI

Didier Grandjean, Univ Geneva, Switzerland


ALPES VAUDOISES
VILLARS-GRYON
DIABLERETS
Oldenhorn
3123m
Quille du Diable
Dôme
3016m
Sommet des Diablerets
3210m
Red Run 8km
Peak Walk by Tissot
Scex Rouge
2971m
GLACIER 3000
Cabane
2525m
Combe d'Audon 7km
Oldenalp
1834m
Black Wall 46°
Oldenegg
1919m
Col du Pillon
1546m
Reusch
lac Retaud
Navette gratuite
Diablerets – Col du Pillon
LES DIABLERETS
1200M
Les Mazots
1717m
Meilleret
1949m
Piste Olympique
Laouissalet
Vers-l'Eglise
1128m
Col de la Croix
1778m
Chaux Ronde
1987m
Bretaye 1805m
lac Noir
lac des Chavonnes
lac de Bretaye
Petit Chamossaire
2034m
Grand
Chamossaire
2120m
Combe
d'Orsay
Roc d'Orsay
2000m
Bouquetins
Golf
La Rasse
Sodoleuvre
Col de Soud
Roches Grises
Barboleuse
VILLARS
1253M
L'Argentine
2422m
Le Coin
2229m
Croix des Chaux
2013m
Solalex
1464m
Les Chaux
1750m
Frience
1540m
Cergnement
Alpe des Chaux
1700m
Pistes faciles
Pistes de difficulté moyenne
Pistes difficiles
Itinéraire
Télécabine
Télésiège
Téléski
Téléphérique
Snowpark
Luge
Boardercross
Fatbike
Ecoles de ski
Secours
Bar d'altitude
Pique nique
Restaurant
Information
Vue panoramique
Parking
Webcam
Ski bus
Train
Zone protection
faune et forêt
GLACIER 3000
P1 Reusch - Oldenegg
P2 Oldenegg - Cabane
P3 Pillon - Cabane
P5 Cabane - Scex Rouge
P6 Oldenalp - Oldenegg
P9 Scex Rouge
P10 Dôme
P11 Quille du Diable
P12 Tsanfleuron
LES DIABLERETS
MEILLERETS
16M Pony - Diablerets
17M Diablerets Express
18M Tapis des Mazots
19M Ruvine
20M Ruvine - Meilleret
21M Laouissalet
22M Vers l'Eglise
26M Conche -
Mi Laouissalet
VILLARS - BRETAYE
A Villars - Bretaye
B Villars - Roc d'Orsay
C Grand Chamossaire
D Bretaye - Orsay
E Combe d'Orsay
F Lac Noir - Chaux Ronde
G Lac Noir - Bretaye
H Lac de Bretaye
J Chaux Ronde
K Petit Chamossaire
L Chavonnes
M Rasse - Chaux Ronde
N Palace
O Tapis Bretaye
GRYON
LES CHAUX
S Barboleuse - Les Chaux
T Sodoleuvre - La Croix
U Gryonne
V Plan Métroz
X Fracherets
Y Ski et luge Frience
Z Tapis Les Chaux
Les lugeurs, VTT, piétons, snow-scoots, etc. ne sont autorisés ni sur les pistes balisées, ni sur les télésièges et téléskis de Villars-Gryon-Diablerets
(exceptés piétons et lugeurs dans la télécabine Diablerets Express 17M)