EWCBR

41th E.W.C.B.R. / E.B.B.S WINTER CONFERENCE

Switzerland, Eurotel, Les Diablerets- March 4th – March 11th, 2023

SUNDAY 5	MONDAY 6	TUESDAY 7	WEDNESDAY 8	THURSDAY 9	FRIDAY 10
	8:00-11:00 #Hal BLUMENFELD *Epilepsy, consciousness & cortical-subcortical networks* 1. Fabrice Bartolomei 2. Cian McCafferty 3. Lim-Anna Sieu 4. Barbara Jobst 5. Hal Blumenfeld + 1hour discussions	8:00-11:00 #Elin LINDSATER *Fatigue on a spectrum: A common symptom domain across disorders?* 1. Anna-Karin Norlin 2. Hege Randi Eriksen 3. Henrik Borsting Jacobsen 4. Silje Reme 5. Hans Knoop 6. Anna Andreasson + 1hour discussions	8:00-11:00 #Wolfram SCHULTZ *Neural economics* 1. Tobias Kalenscher 2. Leo Chi U Seak 3. Simone Ferrari-Toniolo + 1hour discussions	8:00-11:00 #Lina HANSSON *Inflammation & sickness behavior* 1. Lina Hansson 2. Daniel Wilhelms 3. Neil harrison 4. Kristoffer Mansson 5. Marta Zakrzewska + 1hour discussions	8:00-11:00 #Christelle BAUNEZ *When social context modulates cocaine or alcohol effects* 1. Akseli Surakka 2. Lucie Vignal 3. Damiano Terenzi 4. Didier Grandjean + 1hour discussions
17:00-20:00 #Karine GUILLEM *Neurobiological & individuals factors involved in addiction, new targets* 1. Philippe de Timary 2. Christelle Baunez 3. Karine Guillem 4. Mickael Naassila	17:00-20:00 ##Mats LEKANDER *Interdisciplinary models & transdiagnostic symptoms* 1. Harald Engler 2. Sigrid Elsenbruch 3. Mats Lekander 4. Eva Kosek 5. Mariya Ivanovska + 1hour discussions	17h00-20:00 #John AXELSSON *Sleep stress & health* 1. Tanja Lange 2. Stephanie Dimitroff 3. John Axelsson 4. Leonie Balter 5. Sandra Tamm 6. Siri Leknes 7. Simon Kyaga + 1hour discussions	17:00-20:00 #Jérôme BADAUT *A way with words in the name of neurosciences: of terms & tools used to explore the neuronal environment* 1. Jérôme Badaut 2. Simon Cervenka 3. Carole Escartin 4. Lorenz Hirt 5. Jan Pieter Konsman	17h00-20:00 #Lucy PRIVITERA *About but beyond amyloid:latest news on Alzheimer disease* 1. Lucy Privitera, 2. Marco Cambiaghi 3. Emily Hill 4. Christoph Rummel 5. Aniko Korosi + 1hour discussions	17:00-20:00 #Wolfram SCHULTZ *Neural cognition & economics* 1. Peter Bossaerts 2. Jean-Claude Dreher 3. Wolfram Schultz + 1hour discussions
21:00-22:30 Keynote lecture Delphine OUDIETTE 					

The sessions are lasting 3 hours and an additional 1-hour session is opened to discussions to allow brainstorming for collaborative projects.

SYMPOSIUM 1: Neurobiological and individual factors involved in addiction and new therapeutic perspectives

Chair(s) : Mickael Naassila & Karine Guillem
Email: mickael.naassila@u-picardie.fr & karine.guillem@u-bordeaux.fr

Speaker 1: Karine Guillem (CNRS researcher) (INCIA, UMR 5287, Université de Bordeaux)
Prefrontal-based attentional dysfunctions in nicotine addiction
Email: karine.guillem@u-bordeaux.fr

Speaker 2, Philippe de Timary (Professor) (UC Louvain) Microbiota and alcohol addiction
Role of the gut microbiota and feeding behaviour in alcohol. Use disorder : a specific impact on socialization impairments
Email: philippe.detimary@saintluc.uclouvain.be

Speaker 3, Christelle Baunez (CNRS researcher) (INT, Marseille)
Targeting the subthalamic nucleus in addiction: which network is involved?
Email: christelle.baunez@cnrs.fr

Speaker 4, Targeting 5HT2A receptors in alcohol addiction, back to the future
Mickael Naassila (Professor) (Université de Picardie Jules Verne, INSERM)
Email: mickael.naassila@u-picardie.fr

SYMPOSIUM 2: Epilepsy, Consciousness and Cortical-Subcortical Networks

Summary:
Powerful advances in neuroimaging, electrophysiology and computation have provided new insights into mechanisms of normal consciousness and impaired consciousness in neurological disorders. In this symposium, bridging human research and animal models, we will discuss how the same large-scale cortical and subcortical brain networks crucial for normal consciousness are disrupted in epilepsy. Understanding mechanisms of impaired consciousness has major consequences for patient prognosis as well as real world implications including driving safety in people with epilepsy. Emerging data from patients and translational animal models suggest novel therapeutic approaches, such as neurostimulation of deep brain arousal circuits to restore consciousness when it is lost. The speakers will present the latest progress on these topics and will engage the audience in highly interactive participation and discussion.

Agenda:
Human Studies of Impaired Consciousness in Focal Epilepsy
Fabrice Bartolomei, MD, PhD
Decreased Neuronal Firing with Impaired Consciousness in Rat Absence Seizures
Cian McCafferty, PhD
Mouse Focal Limbic Seizures Cause Reduced Cholinergic Arousal and Impaired Consciousness
Lim-Anna Sieu, PhD
Interictal Epileptiform Discharges: Effects on Cognition and Response to Music
Barbara Jobst, MD
Transient Impaired Consciousness: Interictal Epileptiform Discharges and Driving Safety
Heinz Krestel, MD, PhD
Neurostimulation to Restore Consciousness in Temporal Lobe Epilepsy
Hal Blumenfeld, MD, PhD

Faculty:
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SYMPOSIUM 3: Title of the symposium: Interdisciplinary models and transdiagnostic symptoms

Session abstract: Interdisciplinary multilevel theoretical approaches are needed to understand symptoms that are common across disorders, such as pain, stress and fatigue. The session will both discuss theoretical models and obstacles for integrated perspectives regarding body-mind issues such as belief models, language and placebo/nocebo processes. In addition, the session will include recent findings in research that apply psychoneuroimmunological approaches to understand pain disorders, stress responses and fatigue/motivation.

Organizer & Chair : Mats Lekander
Email: mats.lekander@ki.se

Speaker 1
Harald Engler, professor, Institute of Medical Psychology and Behavioral Immunobiology, Essen University Hospital, Germany
The need for animal models in placebo/nocebo research
Email: Harald.Engler@uk-essen.de

Speaker 2
Sigrid Elsenbruch, professor. Dep. of Medical Psychology & Medical Sociology, Ruhr-Universität Bochum, Germany
Nocebo effects, fear and hypervigiliance in interoceptive pain - from mechanisms to application
Email: Sigrid.Elsenbruch@uk-essen.de

Speaker 3
Mats Lekander, professor. Stress Research Institute, Stockholm University and Division of Psychology/Osher Center for Integrative Health, Department of Clinical Neuroscience, Karolinska Institutet, Stockholm, Sweden
Intellectual and clinical consequences of mind-body dualism
Email: mats.lekander@ki.se

Speaker 4
Eva Kosek, professor. Division of Neuro, Departement of Clinical Neuroscience, Karolinska Institutet, Stockholm, Sweden.
Is fibromyalgia an autoimmune disease - results from translational studies
Email: eva.kosek@ki.se

Speaker 5
Mariya Ivanovska, assistant professor. Department of medical microbiology and immunology The Medical University of Plovdiv, Bulgaria
Immunological reactivity under acute and chronic stress
Email: marijaku87@yahoo.com

Speaker 6
Marieke van Der Schaaf, professor, Radboud University Nijmegen, Donders Institute for Brain Cognition and Behaviour, The Netherlands
Fatigue during acute systemic inflammation is associated with reduced mental effort expenditure while mental abilities preserved
Email: marieke.vanderschaaf@donders.ru.nl

SYMPOSIUM 4: Title of the symposium: Fatigue on a spectrum: A common symptom domain across disorders?

Organizer @ Chair: Elin Lindsäter, lic. Psychologist, PhD Karolinska Institutet, Department of Clinical Neuroscience
Email: elin.lindsater@ki.se

Symposium abstract:
Fatigue is a complex and often disabling symptom experience common in both healthy individuals and across a range of acute and chronic conditions/disorders. Traditionally, fatigue has been studied in pipelines of specific conditions (e.g., cancer, stroke, IBS, depression, chronic fatigue syndrome/ME), but an accumulating body of evidence suggests that fatigue might best be conceptualized as a transdiagnostic symptom dimension across disorders. This type of conceptualization could contribute to improved interdisciplinary communication and a more rapid accumulation of knowledge regarding mechanisms of fatigue and the development of efficient treatments. In this symposium we present data on fatigue in different medical conditions and discuss implications of fatigue as a transdiagnostic symptom dimension for new mechanistic models and treatments.

Speaker 1 Anna-Karin Norlin, MD, PhD, Pain and Rehabilitation Center, and Department of Health, Medicine and Caring Sciences, Linköping University, Linköping, Sweden
Title of the talk: Fatigue in irritable bowel syndrome is associated with plasma levels of TNF-α and mesocorticolimbic connectivity
Email: anna-karin.norlin@liu.se

Speaker 2: Hege Randi Eriksen, professor, Western Norway University of Applied Sciences Campus Bergen
Title of the talk: Fatigue and lifestyle factors associated with fatigue among women recently diagnosed with breast cancer compared to age-matched breast cancer free controls.
Email: hege.randi.eriksen@hvl.no

Speaker 3: Henrik Børsting Jacobsen. Clinical specialist in psychology. Ph.D. Mind Body Lab, University of Oslo. Department of Pain management, Oslo University Hospital. CatoSenteret Rehabilitation Center, Son, Norway.
Title of the talk: Breast cancer related fatigue: results from an intervention study
Email: h.b.jacobsen@psykologi.uio.no

Speaker 4: Silje Reme, professor, The Mind Body Lab, Dept. of Psychology, University of Oslo & Dept. of Pain Management and Research, Oslo University Hospital.
Title of the talk: A 3-day intervention for cancer-related fatigue and CFS/ME
Email: s.e.reme@psykologi.uio.no

Speaker 5: Hans Knoop, professor, Amsterdam UMS, University Medical Centers, Netherlands
Title of the talk: Transdiagnostic nature of fatigue and common mechanisms in CBT for fatigue across long term medical conditions
Email: hans.knoop@amsterdamumc.nl

Speaker 6: Anna Andreasson, Ass. Professor, Stockholm University, department of psychology, Sweden
Title of the talk: Fatigue as a possible dimensional symptom domain across disorders: Introduction to the Swedish Fatigue Cohort
Email: anna.andreasson@su.se

SYMPOSIUM 5: Title of the symposium: Sleep, stress and health

Session abstract:
Humans have not only evolved physiological and psychological adaptions to manage and overcome challenges and threats, but also cultural peculiarities such as vaccinations to further boost our ability to defend ourselves from pathogens. The first part of this session focus on how factors (such as adrenalin signaling, psychological factors and sleep loss) predict how effectively our immune system is to defend us from pathogens and how it reacts to vaccinations. This will be followed by presentations of how challenges, such as disturbed sleep, pharmacological treatment and low-grade inflammation, affect cognition and subjective experiences, including potential mechanisms behind worsened mood and depression.

Organizer & Chair: John Axelsson
Email: john.axelsson@ki.se

Speaker 1
Tanja Lange, PhD, Department of Rheumatology & Clinical Immunology | University of Lübeck, Germany
Sleep, adrenergic signaling, and anti-viral immunity
Email: Tanja.Lange@uksh.de
Abstract: Adrenoceptors are key in the body's response to psychosocial stress by regulating, for example, vasoconstriction and vasodilatation. Adrenoceptors also drive immunological changes such as inflammation with increases in leukocyte counts. We assessed levels of catecholamines, as well as numbers and functions of various leukocytes (including virus-specific T cells) to delineate the role of adrenergic signaling in physiological sleep-immune interactions. Our findings point to acute regulatory effects of beta2-adrenoceptor signaling on anti-viral immunity in healthy individuals.

Speaker 2
Stephanie J. Dimitroff, PhD. Neuropsychology, AG Pruessner, University of Konstanz, Germany.
Psychological variables associated the with immune response to COVID-19 vaccination
Email: stephanie.dimitroff@uni-konstanz.de
Abstract: How protected are you against COVID-19 after your vaccination? IgG antibodies are an important part of the artillery for the immune system's defense against the SARS-CoV-2 virus, and its levels are predictive of protection against infection. The number of antibodies produced by some individuals is exponentially higher than others. This difference represents important variance in the future susceptibility to COVID-19 infection. The following study was conducted to determine whether individuals were able to accurately estimate how many antibodies they produced after their COVID-19 vaccinations. Furthermore, past research has linked increased levels of chronic stress, loneliness, and depression with vaccine antibodies. These psychological variables were also measured post-vaccine, to determine if they were related to COVID-19 antibody titers. Methods: 166 participants were recruited to the lab, where a blood sample was taken for analysis. Participants asked to estimate on a scale from 0-10 how many antibodies they produced and were also asked how protected they felt from COVID-19 due to vaccination. They also completed questionnaires assessing levels of chronic stress, depression, and loneliness. Results: Both self-predicted antibody levels, and feelings of protection against COVID-19, were significantly related to their actual IgG spike antibody titers. No relationship between antibody titers and chronic stress, loneliness or depression were found. Results from this study indicate that individuals are able to accurately estimate their IgG titers after COVID-19 vaccination, and demonstrate a robustness of the COVID-19 vaccine immune response against psychological factors like chronic stress, loneliness and depression. Conclusion: These findings suggest individuals who sense they have low protection, probably do. Such information can help individuals make informed choices about self-protective behaviors. Furthermore, psychological variables that have been shown in the past to be related to decreased antibody titers, do not appear to affect the strength of immune response after COVID-19 vaccination.

Speaker 3
John Axelsson, PhD. Stress Research Institute, Stockholm University and Division of Psychology, Department of Clinical Neuroscience, Karolinska Institutet, Stockholm, Sweden.
Sleep loss and inflammatory driven symptoms and behaviour
Email: john.axelsson@ki.se
Abstract: This talk will present a theoretical framework and recent evidence for how sleep loss leads to motivational and behavioural changes. This include: 1) that sleep loss leads to behavioural changes causing the organism to organize behaviours that assure sufficient sleep in an adequate environment, 2) that these behaviours are adaptive and to a large extent overlap with sickness behaviour, and 3) that subjective sleepiness is a likely mechanism driving these behavioural changes. Consequently, insufficient sleep and sleepiness are likely to drive behaviours and symptoms, such as loneliness and a worse lifestyle, problems central in many disorders as well as increasing the risk for developing ill-health. On positive side, improvements of sleep and manipulations of sleepiness could be successful interventions improving motivation aiding a healthy lifestyle and compliance to treatments.

Speaker 4
Leonie Balter, PhD. Stress Research Institute, Stockholm University and Division of Psychology, Department of Clinical Neuroscience, Karolinska Institutet, Stockholm, Sweden.
The effects of low-grade inflammation and sleep loss on model-based and model-free behavioural control: results of an experimental double hit model
Email: leonie.balter@ki.se
Abstract: Behavioural control arises from a balance between model-based and model-free behaviour. Model-based behaviour is cognitively costly but enables adaptation to changes in the environment. In contrast, model-free control is fast, cognitively inexpensive, but inflexible. Overreliance on model-free control and/or reduced model-based control is found across various mental health conditions, suggesting that these modes of control may be influenced by common transdiagnostic processes. Since low-grade inflammation and insufficient sleep are highly common in mental ill-health, we assessed how they by themselves and in combination influence behavioral control.

Speaker 5
Sandra Tamm, PhD. Center for Psychiatry Research, Department of Clinical Neuroscience, Karolinska Institutet, Stockholm, Sweden.
Emotion, affect and mood in insomnia
Email: sandra.tamm@ki.se
Abstract: Experimental studies have suggested that short or disrupted sleep impairs memory consolidation, mood, and perception of emotional stimuli. However, previous studies have been limited by small sample sizes and the use of in-laboratory designs. I will present data on the associations between sleep, overnight emotional and non-emotional memory, emotional perception, and mood in a large, self-selected UK sample.
The associations between poor sleep, insomnia symptoms and mood were strong, whereas the associations between sleep and emotional perception and memory were smaller and non-significant. This is in contrast to published findings where methodical constraints differ

suggesting that care needs to be taken when extrapolating findings from experimental sleep disruption studies to clinical populations measured at home.

Speaker 6
Siri Leknes, PhD, Department of Psychology, University of Oslo, Norway
Sources of variance in subjective experience and behaviour: lessons from psychopharmacology
Email: siri.leknes@psykologi.uio.no
Abstract:

Speaker 7
Simon Kyaga, PhD, Center for Psychiatry Research, Karolinska Institutet, Stockholm, Sweden ; Biogen Inc., Cambridge, MA, USA.
Zuranolone, a Positive Allosteric Modulator of the GABAA Receptor: Hypothesized Mechanism of Action in Major Depressive Disorder
Email: simon.kyaga@biogen.com
Abstract: Depression may result from imbalanced signaling pathways, including deficits in GABA signaling in the brain.1-3 Within the brain, dysregulation of brain activity in regions controlling mood are thought to give rise to depressive symptoms.3 GABA is the primary inhibitory neurotransmitter in the CNS and plays a critical role in maintaining the right balance of inhibition in the brain.4 GABAA receptors are widely expressed in the brain, including in regions dysregulated in depression.5-7 GABA signaling is activated when GABA binds to GABAA receptors.2,8
Zuranolone is an oral, once-daily, 14-day treatment in clinical development for adult patients with major depressive disorder (MDD) or postpartum depression (PPD).9,10 Within the brain, zuranolone is hypothesized to reset brain activity in regions dysregulated in depression.11-16 Zuranolone is hypothesized to bind at a ligand binding site on the GABAA receptors separate from the GABA binding site.8,11,17,18 Upon binding of zuranolone to GABAA receptors, zuranolone rapidly amplifies signaling and expression of various transmembrane GABAA receptors.11

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SYMPOSIUM 6: Neuronal economics

Organizer & Chair Simone Ferrari-Toniolo

- Tobias Kalenscher: Dopamine and social preferences.
- Daniel Hill: Internal reward value coding by dopamine neurons.
- Leo Chi U Seak: Challenging big and small primates with the Independence Axiom of Expected Utility Theory. (junior scientist)
- Simone Ferrari-Toniolo (chair): Reliable economic population signal from unreliable neurons in orbitofrontal cortex.?

Prof Bernhard A. Sabel, SAVIR-Center, Magdeburg & University of Magdeburg, Germany: Massive production of fake science publications by “paper mills”: a thread to the permanent scientific record?

SYMPOSIUM 7: A way with words in the name of neuroscience: Of terms and tools used to explore the neuronal environment

Orgnizer & Chair: Jérôme Badaut

Describing and explaining are among the main activities of science with the former addressing what is the case and the latter dealing with why and how it is the case. Neuroscience, like any science, invents new terms (neologisms) or repurposing words (e.g. metaphors) to describe and explain novel findings. Experimental and clinical scientists sometimes underestimate the importance of framing for guiding research questions and tend to think that terms and notions are superfluous or interchangeable. After a short introduction to these considerations by Dr Konsman, this session will illustrate with three examples, how research findings can be in tension with concepts within basic and clinical neuroscience.

A first example involves the term blood-brain barrier. This metaphor of a barrier has proven important in driving research on brain endothelial cells and glial cells and processes closely associated with blood vessels in the central nervous system. However, the selectivity aspect that was emphasized by the researchers who coined the term blood-brain barrier has often been lost in more recent writings. Dr Badaut will argue that because research findings have corroborated the selectivity of the blood-brain interface, the metaphor of border would be a much more apt one.

A second example concerns the function and behaviour of astrocytes, the most populous glial cells in the brain. Although astrocytes have long been recognized to react to nervous tissue injuries and infection, there has been a proliferation of different terms to describe the

phenotype/function of the cells. In addition, it has been progressively recognized that astrocytes play an important, so-called 'homeostatic,' role in the absence of injury or infection by ion buffering and neurotransmitter recycling. Based on recent findings, Dr Escartin will discuss the need to revise the nomenclature to describe astrocytes activation and reactivity.
The concept of "ischemic penumbra" appeared in the eighties describing a condition of hypoperfusion of the brain, between two thresholds- the upper threshold of electrical failure and the lower of energy and ion pump failure (Astrup & al, Stroke 1981). Since then, the management of acute ischemic stroke patients has improved massively. Although the definition of ischemic penumbra has evolved, the elegant metaphor has prevailed. Dr Hirt will review and discuss the concept of "ischemic penumbra".
Finally, the third example involves the label given to targets of certain radioligands used for PET imaging. The 18 kDa Translocator Protein (TSPO) was initially framed as molecular imaging marker of brain microglial activation, and applied in neurological and psychiatric disorders. However, more recent translational research has shown that the relationship between TSPO and both cell type and activation state is complex. Dr Cervenka will discuss the present state of affairs and make proposals about how to interpret the findings obtained with TSPO-binding radioligands.
Speakers:
Jérôme Badaut (Magnetic Resonance Biol. Syst., CNRS, Univ. Bordeaux, France)
Simon Cervenka (Dept. of Medical Sciences, Psychiatry, Uppsala Univ., Uppsala, and Dept. Clinical neuroscience, Karolinska Inst., Stockholm, Sweden)
Carole Escartin (Lab. Neurodegen. Dis., CNRS-CEA, Univ. Paris Sarclay, France)
Lorenz Hirt (Département de neurosciences cliniques, CHUV et université de Lausanne)
Jan Pieter Konsman (ImmunoConcept, CNRS, Univ. Bordeaux, France)

SYMPOSIUM 8: Title: Inflammation and sickness behavior

Chair: Lina Hansson
Session abstract:
Intruding pathogens will activate immune cascades aiming to neutralize the invader. This defensive response also includes immune-brain signaling that reshape behavior to promote recovery. Such sickness behaviors consist of several symptoms such as anxiety, fatigue, and social withdrawal. Aspects of a sickness response can be detected in others, either by the naked eye or through imaging techniques, allowing the possibility to contain contagion or predict health trajectories. This symposium will cover several aspects of sickness behavior including gait alterations, facial and neural correlates, and inter-individual variabilities. Moreover, the symposium will address different methodological approaches to assess sickness behavior and discuss open science in psychoneuroimmunology.

The walking sick: what predicts the detection of walking sick individuals?
Lina Hansson, PhD-student. Department of Clinical Neuroscience, Karolinska Institutet and Department of Psychology, Stockholm University, Stockholm, Sweden.
The ability to detect sick individuals is crucial for survival, by allowing avoidance of contagion. We have shown that humans can detect sick individuals from facial cues and body odors, but perception of these cues requires close proximity to the infectious person.

Given that gait patterns can be detected from a distance and are altered during sickness, it would be beneficial to detect sickness from biological motion. Here, we investigate if sick individuals can be identified based on solely biological motion and which factors that may predict this detection.

In two studies, raters watched video recordings and point-light displays (i.e. dots depicting the body joints) of walking individuals who were either experimentally sick (after injection with lipopolysaccharide at 2.0 ng/kg bw) or healthy (after a placebo injection). In study 1, 106 raters classified each walking individual as either sick or healthy. In study 2, 106 other raters graded health of the stimuli on a visual analogue scale. We assessed the predicting effect on sickness detection (study 1) and apparent health (study 2) of walking parameters (objective measures of stride length, width, time, as well as knee angle, arm angle, and head angle) and well-known sickness responses (Sickness Questionnaire score, pain intensity, body temperature, and interleukin-6 concentration).

In study 1, the sensitivity was 59% for videos and 57% for point-light displays, while the specificity was 74% for videos and 61% for point-light displays. Shorter steps was the only predictor of the detection of sick individuals from video recordings (β=0.712(0.257), p=0.02). In the point-light displays, slower, wider, stiffer and shorter steps, all predicted a better sickness detection (β=0.0003(0.0001)-0.415(0.126), $p<0.05$).

In study 2, sick walkers were rated as having worse health compared to the same walkers when healthy, both in videos and point-light displays. Lipopolysaccharide-induced slower, shorter and stiffer steps (B=5.214(1.888)-6.385(2.083), $p<0.01$), as well as higher interleukin-6 concentrations (B=0.051(0.020), p=0.01), predicted worse health ratings of sick individuals in the video recordings. In the point-light displays, lipopolysaccharide-induced slower, shorter and stiffer steps, and more head tilting, predicted worse health ratings of sick individuals (B=4.185(1.892)- 6.701(2.092), $p<0.05$).

The results indicate that specific changes in walking parameters may aid in sickness detection, possibly regulating approach-avoidance behaviors towards sick peers.

Peeling Back the Layers: Uncovering Acute Illness with live cutaneous Imaging and Spectroscopy

Daniel Wilhelms, associate professor. Department of Biomedical and Clinical Sciences, Linköping University, Stockholm, Sweden.

Experienced emergency physicians as well as laymen often describe that a quick glance can be sufficient to determine if a person is healthy or not, referring to a certain “look of illness”. Although this has been a recurring theme in medicine, art, and literature for centuries, and most people have a clear internal representation of what this means, there are few objective ways to operationalise this in a clinical healthcare setting. At the same time, there is accumulating evidence to suggest that the “look of illness” may indeed convey relevant, physiological information about the current physiological state as well as the prognosis of a patient.

We seek to determine the physiological underpinnings of this phenomenon by developing and evaluating imaging technology to map changes in cutaneous microcirculation, metabolism and response to standardized provocations. Starting with the classical capillary refill test, I will give a brief overview of the use of novel imaging techniques such as

Reflectance Spectroscopy, Spatial Frequency Domain Imaging (SFDI) and Hyperspectral Imaging (HSI) in the search for an objective representation of the “look of illness”. Inflammation and brain microstructure.
Neil Harrison, professor. Cardiff University Brain Research Imaging Centre , Wales, UK.
Brain structure and sensitivity to inflammatory activation
Kristoffer Månsson, associate professor. Department of Clinical Neuroscience, Karolinska Institutet, Stockholm, Sweden.
The present study aimed to investigate acute effects of endotoxin (i.e., lipopolysaccharide, LPS) exposure on gray matter volume using voxel-based morphometry (VBM) on structural magnetic resonance T1-weighted images. A total of 23 healthy individuals underwent four scans: one session during intravenous LPS exposure (0.8 ng/kg body weight), one during saline placebo, and two baselines without exposure. VBM analysis revealed regional and bilateral significant reductions and increases in gray matter volume (whole-brain voxel-wise, FDR corrected $p < .05$; and support vector machine learning, classification accuracy at 96%, $p = .000006$; AUC = 1) in the anterior insula, amygdala, and orbitofrontal cortex during exposure to LPS, but not placebo. These findings suggest that acute immune challenge can modulate estimated gray matter volume, which may contribute to altered interoceptive processing and immune-related behaviors. Further studies are needed to explore the functional implications of these changes and their potential role in the pathogenesis of immune-related disorders. The present study provides new insights into the complex relationship between the immune system and the brain’s sensitivity, highlighting the importance of studying the neurobiological underpinnings of immune-related behaviors in health and disease.

Open science in psychoneuroimmunology
Marta Zakrzewska, PhD. Department of Clinical Neuroscience, Karolinska Institutet, Stockholm, Sweden.
Reproducibility and replicability, the ability to reconstruct the research methods of prior studies and thus obtain similar results with the same or with new datasets, is a cornerstone of science. Both rely on whether scientists make the research process open with the help of preregistration or by sharing materials, data, and/or analysis pipelines at a later point. In my talk, I will go through recent initiatives to evaluate reproducibility and open science in different fields including psychology, neuroimaging research, and chemosensation. I will use psychoneuroimmunology (PNI) and the official journal of the PNI Research Society (Brain, Behavior, and Immunity) as an example to illustrate how common open science practices were in 2022. I will discuss how reproducibility and open science can be incorporated in order to benefit the robustness of published results and the long-term success of a given field.

SYMPOSIUM 9: About but beyond amyloid: latest news on Alzheimer disease

Alzheimer's Disease (AD) is a neurodegenerative brain disorder characterized by a progressive decline in cognitive functions and reduced autonomy through the deterioration of neuronal cells and their networks. AD disease has been mainly attributed to extracellular accumulations of abnormally folded amyloid β (Aβ) proteins that polymerize and aggregate forming precipitated plaques or circulating soluble oligomers. Given the pathological phenotype, in the last few decades, the research focus has been aggressively targeting amyloid beta for developing disease-modifying therapeutics. Accordingly, Amyloid-clearing drugs would provide an incremental benefit to possibly delay the progression of the degeneration at the best. However, there is still a pressing need to target and understand the onset of Alzheimer's Disease, while contradicting evidence support other etiopathogenic targets, maybe concurring, as possible causes of the AD.
Bringing together a wide range of expertise and giving opportunity to an Early Career Researcher, the session is an opportunity to discuss latest advances on AD research focusing on approaches that do not target solely the amyloid cascades. In this session we will discuss the role of the mGluR-p38-MK2 cascade in synaptic plasticity and memory deficit in a murine model of AD (APP/PS1), the involvement of 5-HT neurons in the cognitive decline occurring during the progression of the disease in 3xTg-AD mice, the identification of relevant tau truncates and their pathophysiological actions in single neuron and possible ways to control neuroinflammation during the progression of the disease. In the end, we will identify early-life risk factors and diet and how they relate with the pathology of Alzheimer's disease using ex vivo / in vivo animal and human cohort data.

Organizer @ Chair : Lucy Privitera
Email: l.privitera@qmul.ac.uk
Speaker 1: Dr Lucy Privitera, Queen Mary University of London, Malta Campus;
Title : mGluR-p38-MK2 cascade as a prime mediator of hippocampal synaptic plasticity and spatial learning deficit in APP/PS1 mice
Email: l.privitera@qmul.ac.uk
Speaker 2: Dr Marco Cambiaghi, University of Verona (IT); Title : Serotonergic system dysfunctions in the 3xTg mice are associated with Alzheimer's Disease progression
Email: marco.cambiaghi@univr.it
Early Career Researcher & Speaker 3: Dr Emily Hill, University of Warwick (UK);
Title : Using TAU truncations to understand the pathophysiological actions of oligomers in single neurons
Email: E.Hill.4@warwick.ac.uk
Speaker 4: Prof. Christoph Rummel, Giessen University (DE);
Title : Deciphering the role of microglia in neuroprotective effects of n-3 polyunsaturated fatty acids and their pro-resolving metabolites using neuro-glial and organotypic hippocampal slice cultures
Email: Christoph.D.Rummel@vetmed.uni-giessen.de
Speaker 5: Dr Aniko Korosi, University of Amsterdam (NL);
Title: Early-life stress as risk factor for Alzheimer's Disease: focus on inflammation and lipid mediators
Email: a.korosi@uva.nl

SYMPOSIUM 10: Title of the symposium: When social contact modulates cocaine or alcohol effects

Organizer : Christelle BAUNEZ
Chair(s) : Christelle BAUNEZ & Didier GRANDJEAN
Email: didier.grandjean@unige.ch

Speaker 1, Title, Institution, Title of the talk:
Akseli Surakka (Junior, PhD student) (CIMH-Manheim; Germany)? The intricacies of choice: When rats prefer alcohol over social interactions
Email: Akseli.Surakka@zi-mannheim.de

Speaker 2, Title, Institution, Title of the talk: Lucie Vignal (Junior, PhD student) (INT, Marseille, F) Role of the subthalamic nucleus in the influence of social presence on cocaine or alcohol intake after escalation
Email: lucie.vignal@univ-amu.fr

Speaker 3, Title, Institution, Title of the talk: Damiano Terenzi (Junior, Post-doc) (INT, Marseille, F) Neural network mediating the influence of the social presence on performance of cocaine users in a stop-task
Email: damiano.terenzi@univ-amu.fr

Speaker 4, Title, Institution, Title of the talk: Didier Grandjean (Univ Geneva, Switzerland) : Neural network mediating the influence of the social presence on performance of alcohol users in a stop-task
Email: Didier.grandjean@unige.ch

SYMPOSIUM 11 : Neural cognition and economics (4 speakers)

Organizer & Chair: Wolfram Schultz

- Peter Bossaerts: Computational complexity in economic choice.
- Predrag Petrovic: Predictive coding in psychosis.
- Jean-Claude Dreher: Neurocomputational mechanisms involved in social decision making.
- Wolfram Schultz (chair): Social reward signals in primate striatum and amygdala