36th European Winter Conference on Brain Research
&
EBBS Winter Conference

Villars sur Ollon, Switzerland

March 5-12, 2016

PROGRAMME

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Session 6 Supported by the Wellcome Trust.

Saturday, March 5 : 17:00 – 19:30

Sunday, March 6 : 16:30 – 19:30

Scientific Registration at the :

EUROTEL Victoria
Villars sur Ollon, Switzerland

Corinne Kanakis, Conference Registration Desk

Monday, March 7, 8 :30 – 8 :45

Opening of the 36th EWCBR&EBBS Winter Conference

Chairman : Christophe BERNARD
President of Promotion des Neurosciences Européennes

SESSION 1
Monday March 7th – 8:30 – 10:30

Neuroprotection : exploring new slopes

Organizers: **Anne-Karine Bouzier-Sore, Luc Pellerin**

Abstract:

There are currently few clinical strategies in place which provide effective neuroprotection and repair for neurological injuries, despite an intense international effort over the past decades. One possible explanation for this is that a deeper understanding is required of how endogenous mechanisms act to confer neuroprotection. This symposium is composed of five talks, for molecular mechanisms to new therapeutic approaches. Luc Pellerin will present *in vitro* that bolstering neuronal energetics might provide an effective neuroprotective strategy. He will describe data showing that LOSAC, a toxin from a Brazilian caterpillar, protects cultured neurons from medium deprivation-induced cell death via an enhancement in the expression of the lactate transporter MCT2. Anne-Karine Bouzier-Sore will show that lactate can act as a neuroprotective agent in neonate rats after hypoxic-ischemic injury. She will detail her results showing that an intra-pertoneal lactate injection performed 30 minutes after the insult reduced the lesion size and cytotoxic oedema as revealed by diffusion MRI. Jérôme Badaut will present data showing that the JNK inhibitor D-JNKI1 permits improvement of motor and cognitive functions when administered 3 hours post-insult in a juvenile rat model of traumatic brain barrier and enhanced vascular perfusion. Lorenz Hirt will discuss recently published results showing that bot L-and D-lactate injected intravenously or intra cerebro ventricularly offer neuroprotection in a transient middle cerebral artery occlusion (MCAO) mouse model . Evidence suggests that such an effect occurs via an activation of a lactate receptor recently described in the CNS as well as by their intracellular metabolism. Finally, André Obenaus will show that docosahexaenoic acid administered 3 hours after stroke induction reduces the lesion size and improves motor function in a MCAO female rat model. He will also demonstrate by high field MRI that the effect on the lesion size is due to decreased volume of the penumbra. Overall, we hope that this symposium will foster discussions around the necessity to explore new neuroprotective strategies.

Speakers:

- Jérôme Badaut, INCIA, Bordeaux: Neurovascular protection using JNK inhibitor in early brain injury
- **Anne-Karine Bouzier-Sore**, RMSB, Bordeaux : Lactate : more than a neuronal energetic substrate
- **André Obenaus**, Loma Linda Univ, USA :Docosahexaenoic acid treatment rescues the ischemic penumbra after experimental stroke
- **Lorenz Hirt**, CHUV, Lausanne : Lactate and neuroprotection in cerebral Ischemia

SESSION 2
Monday March 7th – 16:30 – 19:30

Inflammation and Behavior

Organizer: **John Axelsson**

Abstract

A better knowledge of neuroimmune interactions during illness is central for our understanding of debilitating symptoms and the ability to develop successful treatments. It is well known that sickness is accompanied by behaviours such as fatigue, fever, altered sleep and food preferences, pain, anhedonia and social withdrawal. These coordinated set of behaviours are often referred to as 'sickness behaviours', and believed to be highly adaptive and aid survival. However, while many of these behavioural changes are favourable acutely, they also cause a lot of suffering, particularly in patients with chronic illnesses. Modern health care has much to gain from a better understanding of neuroimmune interactions, and how immune activation and medication alters brain functioning during illness and treatments. Three of the involved speakers will review and present recent findings of these issues. In addition, the session will also cover how humans change overt behaviours during acute sickness and how these behaviours affect the interactions with surrounding peers.

Speakers:

- **Predrag Petrovic**, Karolinska Institute, Sweden: „Emotional regulation in health and disease"
- **Neil Harrison**, Univ. of Sussex, U.K.: „Intrinsic brain connectivity after interferon (IFN) treatment/Effects of minocycline human memory/amygdala reactivity and depression after IFN treatment/effects of anti-TNF on mood and fatigue in RA patients"
- **Cobi Heijnen,** Univ. of Texas, USA: „Chemo brain and ways to prevent it"
- **John Axelsson,** Karolinska Institute, Sweden: „Overt behavioural changes in sick humans"
- **Mats Lekander**, Karolinska Institute, Sweden: „Disease avoidance in health anxiety"

SESSION 3
Tuesday March 8th – 8:30 – 10:30

New data in the addiction field : from neurobiology to therapeutic interventions

Organizer: **Mickael Naassila**

Abstract:

The symposium will present recent and new data on the vulnerability to alcohol and cocaine addiction. A therapeutic perspective will be also presented using either pharmacological or brain stimulation approaches.

Speakers:

- **Mickael Degoulet**, CNRS & AMU, Marseille, France : "Acute and chronic effects of deep brain stimulation on cocaine escalation in rats"
- **Marcello Solinas**, CNRS & Univ. Poitiers, Poitiers, France: „"Statins as new medications to reduce the Risks of Relapse to Addiction"
- **Sébastien Carnicella**, INSERM & Univ. Grenoble, Grenoble, France : „Role of hypo dopaminergic state in the vulnerability to drink alcohol in rats"
- **Mickael Naassila,** INSERM &Univ Picardie, Amiens, France : „Effects of the current treatments of alcoholism in a new model of alcohol binge drinking in rats"

SESSION 4
Tuesday March 8th – 16:30 – 19:30

"Pain and Inflammation"

Organizer : **Mats Lekander,** Karolinska Institute

Abstract:

In understanding pain regulatory mechanisms, both peripheral and central inflammatory mechanisms have been increasingly investigated during recent years. During inflammatory activation, local and systemic processes affect peripheral nerve endings and central pain regulatory systems in concert, resulting in increased pain sensitivity and spontaneous pain. Such phenomena can be studied with experimental inflammatory provocations in animals as well as in humans, but converging data also stem from studies of inflammatory processes in patients suffering from chronic pain. The session will review findings, present data and discuss methods used to study the relation between inflammation and pain in health and disease, including methods to investigate inflammatory activation in the human brain. In addition, the session will present recent findings and concepts regarding pain regulation and learning outside conscious processing, as well as the role of inflammation in problems that often co-occur with chronic pain.

Speakers

Bianka Karshikoff, Stockholm University, Sweden, bianka.karshikoff@su.se**:"**Sickness hurts: Pain sensitivity during LPS-stimulation in humans"

Eva Kosek, Karolinska Institute, Sweden, Eva.Kosek@ki.se: "Central cytokines and pain"

Karin Jensen, Karolinska Institute, Sweden, Karin.Jensen@ki.se: "Pain processing during sleep"

Annemieke Kavelaars, MD Anderson, Texas, AKavelaars@mdanderson.org:**"**Neuroimmune mechanisms of comorbid pain and depression"

Niels Eijkelkamp, UMC Ultrecht, Netherlands:"The mitochondrial methyltransferase FAM173b promotes chronic pain development"

PLENARY SESSION
Tuesday March 8th – 21:30 – 22:30

« The economic utility signal of dopamine neurons »

Wolfram Schultz
Department of Physiology, Development& Neuroscience, University of Cambridge, Cambridge, UK

Abstract:

Rewards induce learning (positive reinforcement), approach behaviour, economic decisions and positive emotions (pleasure, desire). We investigate basic neuronal reward signals during learning and decision-making, using behavioural and neurophysiological methods.

The phasic dopamine reward prediction error signal is composed of two components, resembling two-component responses in main sensory neurons. The early dopamine response component detects events in discriminately and is influenced by physical impact, novelty, reward generalisation and reward context. The second component codes reward value. Although the first component detects punishers by their physical impact, none of the components shows an activation that reflects the aversive nature of punishers.

Our behavioural studies reveal that monkeys are risk seeking with small rewards and risk neutral and then risk avoiders with larger rewards. The animals' choices are meaningful in satisfying first, second and third order stochastic dominance, which defines rational choices governed by value, symmetric (variance) risk and skewness risk, respectively. The forms of the behavioural utility functional low us to assess the relationship of dopamine predictioner corresponses to economic utility. Indeed, the dopamine responses satisfy first- and second-orders to chastic dominance and code utility. These data unite concepts from animal learning theory and economic decision theory at the level of single reward neurons.

SESSION 5
Wednesday March 9th – 8:30 – 10:30

Mechanistic relevance of deep brain stimulation targets for obsessive compulsive disorder.

Organizers: Christelle Baunez, Institute of Neurosciences La Timone, CNRS & AMU, Marseille, France & **Valérie Voon**, University of Cambridge, Cambridge, United Kingdom

Abstract:
Optimizing neuromodulation in psychiatric disorders requires a careful mechanistic understanding of the neural target and relevance to the neurocognitive network of the psychiatric disorder. This symposium focuses on a translational understanding of the mechanisms underlying neural targets and networks relevant to obsessive compulsive disorder (OCD) and suggests possible relevance to disorders of addiction. The role of optogenetic stimulation of the lateral orbitofrontal cortex in a Sapap 3 rodent mouse model of OCD with excessive grooming on inhibitory impairment in a delay conditioning paradigm and striatal projection activity will be discussed. Manipulation of the subthalamic nucleus (STN) in rodents and its relevance to decision making, loss chasing and disorders of addiction will be covered. The relevance of the STN network on specific forms of impulse control, particularly waiting and reflection impulsivity in humans, will be presented focusing on resting state connectivity, STN stimulation and dimensional relevance across alcohol misuse. The efficacy of deep brain stimulation and dimensional relevance across alcohol misuse. The efficacy of deep brain stimulation (DBS) targeting the STN in OCD and physiological markers of response inhibition will be presented. Finally, efficacy of DBS targeting the nucleus accumbens in OCD and the influence on dopamine release and functional connectivity will be discussed.

Speakers :

- **Eric Burguiere**, ICM, INSERM & UPMC, Paris, France: "Optogenetic probing of cortico-basal loops in compulsive behaviors"
- **Christelle Baunez**, Institut de Neurosciences de la Timone, CNRS & Aix-Marseille Université, Marseille, France: “The subthalamic nucleus (STN) in the control of inhibition: relevance to decision making, loss chasing and addiction“
- **Valérie Voon**, University of Cambridge, Cambridge, U.K.: “The STN network on impulse control in humans”
- **Mircea Polosan**, University of Grenoble, Grenoble, France: “Deep brain stimulation (DBS) targeting the STN in OCD and physiological markers of response inhibition”
- **Martijn Figee**, University of Amsterdam, Amsterdam, The Netherlands: “Deep Brain Stimulation of the Nucleus Accumbens in Obsessive Compulsive Disorders”

SESSION 6
Wednesday March 9th – 16:30 – 19:30

Cell junctions, Connexin and Pannexins in the Brain : New Targets for Therapeutics

Co-Chairs : **Christian Naus,** Univ. of British Columbia**, and Luc Leybaert,** Univ. of Ghent, Belgium

Abstract :

Cells within the CNS communicate at many levels involving processes ranging from neuronal synaptic interactions, astrocyte networks, gliovascular regulation, inflammatory microglia and blood brain in their high expression level of junctional proteins. These include connexins which form gap junction channels that provide the basis for a unique direct cell-to-cell communication, as well as uptake and release of metabolites. More recently, another family of gap junction proteins has been identified, termed pannexins; these proteins share similar membrane topology but no sequence homology with connexins. They also form hexameric membrane channels with a pharmacology somewhat overlapping with connexin hemichannels. Tight junctions are a critical component of the blood brain barrier, with claudins as essential components. In this workshop, we discuss how gap junctions, hemichannels and pannexin channels, and tight junctions may function in brain health and disease. A common theme for each talk will be potential therapeutic applications targeting these junctional proteins in the CNS.

Speakers:

- **Luc Leybaert**, University of Ghent, Belgium: “Radiation by stander effects mediated by brain endo the lialgap junctions”
- **Christian Giaume**, College de France, Paris: “Gap junctions as a therapeutic target in Alzheimer’s Disease”
- **Moises Freitas-Andrade**, University of British Columbia, Canada: “Pannexin channel activation in stroke and neuro degeneration”
- **Michael Koval**, Emory University School of Medicine, Atlanta, Georgia, USA: "Roles for claudine modeling in regulating tight junction permeability"
- **Christian Naus,** University of British Columbia, Vancouver, Canada: “Connexins in the brain facilitate glioma invasion”
- **Dale Laird**, Western University, London, Canada
- “The role of large-pore pannexin and connexin channels in neurosensory hearing-loss"

Wednesday March 9th – 21:00 – 22:30

POSTER SESSION

SESSION 7
Thursday March 10th – 8:30 – 10:30

"Pain mediators and Mechanisms"

Organizer :**Stephen B McMahon**,
Neurorestoration group, King's College, London, UK

Abstract :

In vivo imaging of nociceptive sensory and spinal neuron activity in the mouse
The conventional way to monitor neuronal activity is electrophysiological, in which the activity of single or groups of neurones is sampled. Recording electrode arrays allow monitoring at multiple sites and of multiple neurones. An alternative approach is now becoming more widely adopted and this is to use genetically encodable calcium sensitive dyes which can be expressed in cells of interest by surgical or genetic methods. In this talk I will review how we have applied these techniques to the study of pain in mice.
We have used two commercially available tools. In the first, a 'floxed-stop' GCaMP6s sequence is expressed in all cells of the mouse at the Rosa locus. Cells which express Cre-recombinase then synthesise GCaMP6s protein, which is a calcium sensitive dye. This mouse is available from Jackson laboratories. The second approach is to use AAV9 vectors which express GCaMP6s in the cells they transfect. We have found that intrathecal injections or 5ul of AAV9 will transfect most sensory neurones in the DRG. GCaMP6 protein is expressed in sensory neurones after about 2 weeks. If small volumes of AAV9 are microinjected into CNS parenchyma, they transduce neurones preferentially. AAV9, but not AAV1 or 2 serotpyes, have the property of retrograde transport in long projection neurones. We have injected this AAV9 vector into the parabrachial nucleus of rats and mice and this leads to GCaMP6 expression in many lamina I projection neurones in the spinal cord. In anaesthetised rodents, we can image with confocal or multiphoton microscopy the activity-induced fluorescence of primary sensory and spinal lamina I projection neurones to a range of peripheral stimuli. This talk will review the advantages and limits of this technique and illustrate its use to study pain.

Supported by the Wellcome Trust.

Speakers :

- **Felix Viana**, Instituto de Neurociencias, Alicante "Molecular physiology of mammalian cold thermo receptors"
- **Natalia Malek,** Institute of Pharmacology PAS, Krakow : „Characterization of chronic pain development in the animal model of osteoarthritis"
- **Steve McMahon**, Kings College, London: "Novel pain mediators in BladderPain Syndrome"
- **Jiri Palacek** : Center of Biomedical Research, Czech republic: "TRPV1 receptors in pain modulation"
- **Peter Reeh**: Institut für Physiologie & Pathophysiologie, Erlangen Univ."Light is painfully blue for TRPA1"
- **Jordi Serra**: Univ. of Barcelona "Common C-fiberab normalities in diverse painful conditions: recent findings"

SESSION 8
Thursday March 10th – 16:30 – 19:30

"Mechanisms underlying headhache, migraine and epilepsy"

Organizer **:Karl Messlinger,** Karolinska Institute, Stockholm, Sweden

Abstract :

Primary headaches including migraine are believed to depend critically on dysregulation of the peripheral and central nervous system and may therefore share similarities with pathological mechanisms involved in epilepsy and pain. The session will be a platform to discuss genetic, molecular, neurophysiological and behavioral aspects of increased excitability in cellular and physiological models of migraine and epilepsy and to probe the usefulness of these models for the understanding of clinical features of these diseases. The topic of the session links closely to the EU project EUROHEADPAIN, which started in early 2014. Research laboratories and clinical departments of several European countries collaborate in this project to advance the understanding of the pathogenesis of headache and migraine, to study the mechanisms underlying its transition into chronic states and to probe new ideas for its therapy.

Speakers:

Antoinette Maassen Van den Brink, Dpt of Internal Medicine, Rotterdam, Netherlands, „Neuro vascular mechanisms involved in migraine"
Arn M.J.M. Van den Maagdenberg, Leiden University Medical Centre, Leiden, Netherlands ; „Investigating genes and mechanisms of migraine using transgenic mouse models"
Else A. Tolner, Leiden University Medical Centre, Dpt of Human Genetics, Leiden, Netherland : „Functional studies tounravel shared mechanisms in migraine and epilepsy"
Sandra Vilotti, Neurobiology Sector, Internat. School for Advanced Studies, Trieste, Italy: „Selective up regulation of P2X3 receptors under liessen sitization of trigeminal sensory neurons"
Karl Messlinger, Institut für Physiologie & Pathophysiologie, Univ. Erlangen-Nürnberg, Erlangen, Germany: „Sources and actions of CGRP in trigemino vascular and visceral systems"

SESSION 9
Friday March 11th – 8:30 – 10:30

"Epilepsy and related morbidities"

Organizer :**Claude Wasterlain,** UCLA

Abstract :

Epilepsies are the second most common neurological disorder after migraine, with multiple causes and treatments. In addition, they are often associated with comorbidities (cognitive deficits and depression), increasing their societal cost. Epilepsies are drug-resistant in 30% of the cases. It is therefore important to understand the causal elements leading to epilepsy and comorbidities, as well as external factors that may influence their occurrence, in order to propose better therapeutical solutions. This session will show that past stressful experiences can influence the development of epilepsy and comorbidities. We will discuss the role of temperature and stroke, the two most common causal factors that can lead to epilepsy in the developing and adult brain, respectively. Finally, we will provide a translational perspective looking at the future of imaging techniques for the diagnosis of epilepsies.

Speakers:

Christophe Bernard, Institut de Neuroscience des Systèmes, Marseille : " The diathesis-epilepsy model, or how past events influence the occurrence of epilepsy and comorbidities"

Olaf Paulson, Rigshospitalet, Copenhagen, Denmark : "Is there a future role for molecular imaging using PET and SPECT in focal epilepsy and in the presurgical evaluation process ? "

Claude Wasterlain, UCLA Brain Research Institute: "Seizure-associated neuronal injury in the immature brain :role of temperature"

Denny Fujikawa :UCLA Brain Research Institute" The Neurobiology of Stroke"

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