BRAIN& MIND SYMPOSIUM

OCTOBER 14 - 15, 2021

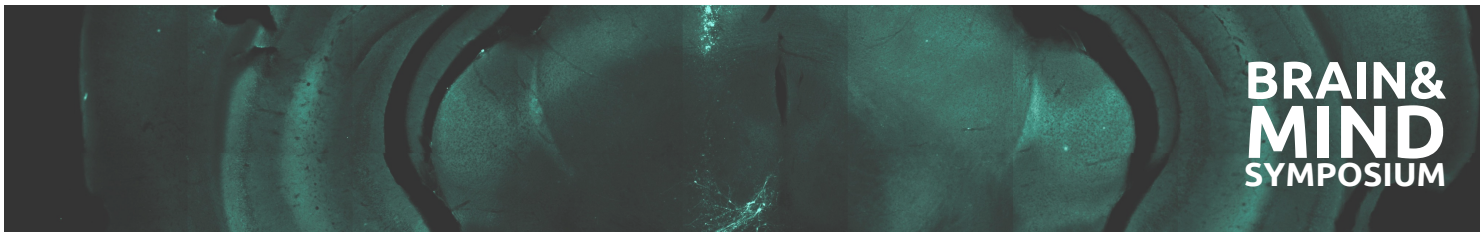
ONLINE

SYMPOSIUM BOOKLET

UNIVERSITY OF HELSINKI

B&M Doctoral Program Brain & Mind

A! Aalto University



Dear Symposium participant,

Welcome to the Brain & Mind Symposium 2021! This symposium is the annual scientific event organised by the doctoral students of the Doctoral Programme Brain & Mind at the University of Helsinki and Aalto University.

In this booklet you will find the final event programme and the short biosketches of all the main speakers as well as the abstracts for the posters presented during the event.

We wholeheartedly welcome you to the Symposium.

Brain & Mind Student Council 2021

Lauri Elsilä
Vicky Gkini
Natali Martyniuk
David Micinski
Annika Schäfer

PROGRAMME

Thursday October 14th, 2021

09.45 – 10.00 **Opening words** by Lauri Parkkonen

10.00 – 12.00 **Symposium: Learning and Memory**

- Gisella Vetere: “Decoding memory formation and stabilization in mice”
- Athena Akrami: “The lingering past in working memory — from sensory history to optimal learning of temporal regularities”

13.00 – 14.00 **Poster presentations**

14.15 – 16.15 **Symposium: Pain and nociception**

- Ewan St. John Smith: “Naked Nociception: Tales from the naked mole-rat”
- Katrin Schrenk-Siemens: “Human stem cell derived neurons: a step towards translational pain research?”

16.30 – 18.00 **Newly established group leaders in Helsinki**

- Teppo Särkämö (University of Helsinki)
- Henrike Hartung (University of Helsinki)
- Coralie Di Scala (University of Helsinki)
- Yoni Levy (Aalto University)
- Olli Pietilä (University of Helsinki)
- Teemu Aitta-aho (University of Helsinki)
- Tom McWilliams (University of Helsinki)

18.45 – 00.00 **Dinner** at GLO Hotel Art

All times in Helsinki time (EEST, UTC+3)

PROGRAMME

Friday October 15th, 2021

9.45 – 12.45 **Symposium: Regulation of motivation & emotions**

- Ewelina Knapska: “Second-hand emotions. What can rodents learn about the world from other’s bliss and fear?”
- Angela Roberts: “Prefrontal regulation of positive and negative emotions in a primate”
- Roshan Cools: “Chemistry of the adaptive mind: lessons from dopamine”

13.45 – 14.45 **Workshop: Climate and environment crisis in neuroscience**

- Kate Jeffery
- Commentary by UH vice-rector Tom Böhling

15.00 – 16.00 **Poster presentations**

16.15 – 18.15 **Symposium: Glial cells**

- David Attwell: “The role of glia and pericytes in regulating brain blood flow in health and disease”
- Benedikt Berninger: “Engineering new interneurons in the cerebral cortex in vivo”

18.15 **Closing words** by Iiris Hovatta

All times in Helsinki time (EEST, UTC+3)

SYMPOSIA SPEAKERS

Gisella Vetere

"Decoding memory formation and stabilization in mice"

Gisella Vetere is a Professor in Neurobiology and the team leader of the C4 group (Cerebral codes and circuits connectivity) at the ESPCI in Paris since August 2018. Her training is in behavioural, cellular and systems neuroscience, with a specific focus on learning and memory. She did her PhD in Rome (Italy) where she studied synaptic plasticity changes that occur following acquisition of new and long-lasting memories. In 2014 she moved to Canada to work at the Sick Children Hospital in the laboratory of Dr. Paul Frankland. She established the role of structural and functional synaptic changes in the process of memory consolidation. She also artificially engineered specific neurons to create a memory without real experience. She was awarded several prizes and grant for her research including the ANR JCJC, ATIP/Avenir, NARSAD Yonge Investigators, Aspirational Neuroscience Prize and the Brain Star Award.

Athena Akrami

"The lingering past in working memory — from sensory history to optimal learning of temporal regularities"

Athena joined the faculty at Sainsbury Wellcome Centre (SWC), UCL, in November 2018. She obtained her BA in Biomedical Engineering from Tehran Polytechnic (Amirkabir University of Technology) and her PhD in Computational Neuroscience from International School for Advanced Studies (SISSA), with Alessandro Treves. She then became a postdoctoral fellow at SISSA where she switched gears towards experimental neuroscience with Mathew Diamond, and then at Princeton University where she was a Howard Hughes Medical Institute fellow and worked with Carlos Brody on Parametric Working Memory. Her Learning, Inference & Memory laboratory at SWC, is focused on understanding the fundamental principles of statistical learning – the ability of the brain to discover and exploit relevant regularities and structures in the world in an unsupervised manner. In all of her research programs, experiments are intertwined with hypotheses drawn from theoretical investigations and computational modelings. Since April 2020, Athena has also become an accidental advocate and researcher of Long COVID.

SYMPOSIA SPEAKERS

Ewan St. John Smith

"Naked Nociception: Tales From The Naked Mole-Rat"

Ewan completed his undergraduate degree in pharmacology at the University of Bath, followed by a PhD with Peter McNaughton at the University of Cambridge working on acid-sensing ion channels. He then moved to work with Gary Lewin at the Max-Delbrück Centre in Berlin as an Alexander von Humboldt Research Fellow, where he began working on pain peculiarities of the naked mole-rat. This was followed by a 1-year stint with Niels Ringstad at the Skirball Institute of Biomolecular Medicine at NYU as a Max Kade Foundation Fellow, where he worked on CO2-sensing in C. elegans. In 2013 he was appointed to a Lectureship in Pharmacology at the University of Cambridge where his research group focuses on understanding the molecular basis of nociception using both mice and naked mole-rats as model systems, as well as investigating the cancer resistance and healthy ageing of naked mole-rats. He was promoted to Senior Lecturer in 2017 and Reader in 2019, also being a Fellow of Corpus Christi College where he is Director of Studies in Biological Natural Sciences. Work in the Smith lab is funded by the BBSRC, MRC, Versus Arthritis, Dunhill Medical Trust, Astra Zeneca, Beiersdorf and GSK.

Katrin Schrenk-Siemens

"Human stem cell derived neurons: a step towards translational pain research?"

Katrin has been working with human embryonic stem cells for 20 years, with an emphasis on the generation of central and peripheral neurons. She studied Biology, received her PhD in the field of neuroscience from the University of Basel, Switzerland and worked with adult stem cells during her first postdoc at Stanford University. She then started to focus on the development of differentiation procedures to generate sensory neurons during her second postdoc in Berlin. Currently Katrin is located at the Institute of Pharmacology at Heidelberg University and a co-PI in the Heidelberg pain consortium (SFB1158). Her main interest is the development of an in vitro model system using human pluripotent stem cell-derived nociceptors and glutamatergic neurons as a novel tool to investigate synaptic transmission in models of sensitization or pain-related mutations.

SYMPOSIA SPEAKERS

Ewelina Knapska

"Second-hand emotions. What can rodents learn about the world from other's bliss and fear?"

Born in Poland in 1977, Ewelina Knapska obtained MSc degrees in biology and psychology from the University of Warsaw. In 2001 she started working with Tomasz Werka and Leszek Kaczmarek, studying the heterogeneity of the amygdala in control of positive and negative emotions. After obtaining her Ph.D. from the Nencki Institute of Experimental Biology PAS in 2006, Ewelina moved to Ann Arbor, USA, for a postdoc in the laboratory of Prof. Stephen Maren at the University of Michigan. She moved back to Poland in 2008, getting a stipend from the Foundation for Polish Science and becoming an assistant professor at the Nencki Institute of Experimental Biology PAS. In 2012, she obtained habilitation (DSc) and became an associate professor and the head of the newly created Laboratory of Neurobiology of Emotions. In 2016 she received a Starting Grant from the European Research Council to study the amygdala's role in controlling socially transferred emotions. Since 2018 she has been Vice-President of the Centre of Excellence for Neural Plasticity and Brain Disorders (BRAINCITY) in the Nencki Institute of Experimental Biology.

Angela Roberts

"Prefrontal regulation of positive and negative emotions in a primate"

Angela Roberts obtained her PhD in neuroendocrinology from University of Cambridge (1985) and following postdoctoral studies researching into the neural and neurochemical basis of cognitive flexibility was appointed Lecturer in Department of Anatomy, Cambridge, in 1996, becoming Professor of Behavioural Neuroscience in 2010. She was elected a Fellow of the Academy of Medical Sciences in 2016. Recent scientific contributions have involved establishing non-human primate models of positive and negative emotion regulation, fractionating out the distinct prefrontal networks that may underlie the varied aetiology of affective disorders and elucidating the sensitivity of these networks to anxiolytics/antidepressants essential for the more effective targeting of current pharmacotherapies. She received the Goldman-Rakic Prize for outstanding achievements in Cognitive Neuroscience in 2020.

SYMPOSIA SPEAKERS

Roshan Cools

"Chemistry of the adaptive mind: lessons from dopamine"

Roshan Cools has made lasting contributions to cognitive neuroscience, providing empirical foundations of current theories of dopamine and serotonin's roles in human motivational and cognitive control. She has pioneered the combination of advanced techniques, including pharmacological fMRI, chemical PET, psychopharmacology and computational cognitive science. She is an elected member of the Royal Netherlands Academy of Arts and Sciences and the Academia Europaea. She is also an active member of the Advisory Council for Science, Technology and Innovation (to the Dutch government).

After completing her undergraduate degree in Experimental Psychology at the University of Groningen in 1998, she moved to Trevor Robbins' lab at the University of Cambridge, UK, for a PhD degree (2002), Royal Society Research Fellowships until 2007. She also spent two visiting post-doc years at UC Berkeley working with Mark D'Esposito from 2003. In November 2007 she returned to The Netherlands, where she is Principal Investigator at the Donders Institute for Brain, Cognition and Behaviour and full Professor of Cognitive Neuropsychiatry at the Radboud university medical center in Nijmegen, running a productive group of international researchers studying the chemical neuromodulation of human cognition (www.roshancools.com).

Kate Jeffery

Workshop on the climate crisis

Kate Jeffery is a behavioural neuroscientist at University College London. Her scientific research explores how the brain makes an internal map of space for use in navigation and memory. At UCL she heads the Institute of Behavioural Neuroscience in the Division of Psychology and Language Sciences, and is Vice Dean (Research) for the Faculty of Brain Sciences. She is also co-director of the electrophysiology company Axona Ltd, which makes high-density recording systems for behavioural neuroscientists, and is a Fellow of the Royal Society of Biology and Fellow of the Royal Institute of Navigation, where she chairs the Cognitive Navigation Special Interest group. She is interested in enhancing public understanding of science and in this role, has given many talks not only about her research but also about the climate crisis.

SYMPOSIA SPEAKERS

David Attwell

"The role of glia and pericytes in regulating brain blood flow in health and disease"

David Attwell did a first degree in physics and a PhD on the electrophysiology of nerve and muscle cells with Julian Jack in Oxford, before spending 2 years in Berkeley studying the retina with Frank Werblin. On returning to the UK, he moved to the Department of Physiology at University College London, where he has remained ever since. He has worked on a wide range of subjects including the properties of glial cells, glutamate transporters, stroke, the formation of myelin by oligodendrocytes, how neuronal computation is powered and the control of cerebral blood flow. He was made a Fellow of the Royal Society in 2001.

Benedikt Berninger

"Engineering new interneurons in the cerebral cortex in vivo"

Benedikt studied biology at the University of Munich. After a PhD on activity-dependent neurotrophin regulation in the laboratory of Hans Thoenen, he joined Mu-ming Poo's lab at the University of California San Diego to investigate rapid action of neurotrophins at synapses and growth cones. Following his return to Munich he got fascinated by adult neural stem cells and the neurogenic fate potential of glia. Working with Magdalena Götz at the Stem Cell Institute, Helmholtz Zentrum München and the University of Munich, he discovered the proneural factors Neurog2 and Ascl1 as key reprogramming factors for glia-to-neuron conversion. In 2012 he was appointed professor of Physiological Chemistry at the University Medical Center Mainz and in 2018 he became Professor for Developmental Neurobiology at King's College London. Since 2021, Benedikt leads also a satellite group at the Francis Crick Institute. His research covers a broad range of topics from molecular mechanisms of neuronal reprogramming to circuit remodelling.

NEWLY ESTABLISHED GROUP LEADERS

Teppo Särkämö

Teppo Särkämö is an Associate Professor of Neuropsychology at the Department of Psychology and Logopedics, University of Helsinki and the Group leader of the Music, Ageing and Rehabilitation Team (MART) at the Cognitive Brain Research Unit (CBRU). His research focuses on the psychological and neural mechanisms of language and music and their neurological deficits (aphasia and amusia) and on the effects of musical activities and music-based rehabilitation methods in ageing and in different neurological illnesses. Currently, he leads a large ERC-funded project on singing in normal ageing and aphasia and on music-evoked memories in Alzheimer's disease.

Henrike Hartung

Henrike Hartung is an Academy of Finland Research Fellow and associated group leader at the Neuroscience Center, Helsinki Institute of Life Science (HiLIFE), University of Helsinki. Her main research interest is on the developmental origin of neuropsychiatric disorders. With her team, she investigates the role of early-life stress, including birth stress, on the functional development of the brain with a focus on the serotonergic system and prefrontal-amygdala networks. Her experimental approach mainly includes in vivo state-of-the-art electrophysiological recording techniques in developing rodents in combination with anatomical, pharmacological and behavioural methods.

Coralie Di Scala

Coralie Di Scala got a PhD from Aix-Marseille University (France) in Molecular Neurosciences (supervised by Prof. Fantini and Dr. Chahinian). She discovered a new mechanism of beta-amyloid peptide toxicity within the membrane of cells. In 2018, she joined the Rivera lab (INSERM, France) and focused on stability of membrane proteins within the membrane in a context of epilepsy. In 2019, she won the prize Valérie Chamaillard from the Fondation de France that rewards the best young researcher project on epilepsy in France. In 2020, she started her group at Neuroscience Center as Academy Fellow. Her team deciphers the molecular mechanisms underlying lipid-protein interactions and evaluates their functional impacts for nervous system diseases with an emphasis on epilepsy.

Yoni Levy

Yoni (Jonathan) Levy is a social neuroscientist at Aalto University and in IDC Herzliya. What drives his work in science is the passion to investigate phenomena from multiple perspectives, and merging them into novel representations. In this brief talk he will present his neuroscientific work on timely social phenomena.

NEWLY ESTABLISHED GROUP LEADERS

Olli Pietiläinen

Olli Pietiläinen, Ph.D., is a group leader at the HiLIFE Neuroscience Center (NC) at the University of Helsinki and a researcher at the Stanley Center for Psychiatric Research at the Broad Institute of MIT and Harvard. His group in NC focuses on using genomic approaches in iPSC derived neuronal models to study psychiatric disorders. Olli graduated as Ph.D. from the Institute for Molecular Medicine Finland (supervised by Prof. Aarno Palotie & Academician Leena Palotie). Before joining NC, Olli worked with Professor Kevin Eggan at Harvard University. His work there focused on genomic modeling of genetic risk variants in schizophrenia in iPSC derived neuronal models.

Teemu Aitta-aho

Teemu Aitta-aho is currently a group leader and a lecturer teaching pharmacology and neuroscience at the University of Helsinki, Finland. He studied pharmacy and pharmacology at the Universities of Helsinki and Gothenburg, completed PhD on drug-induced neuroplasticity in 2012, and received postdoctoral training in 2014-2016 in the University of Cambridge. Currently he is interested in neurochemically-defined neurocircuitries in food intake and related motivated behaviours. To understand these, he is using novel cell-type specific artificial receptor-ligand systems and light-guided neural control with optogenetics, as well as brain cell imaging in freely moving animals.

Tom McWilliams

Tom McWilliams has a multidisciplinary training background with expertise in developmental and degeneration neuroscience. He trained with Alun M. Davies and Stephen B. Dunnett on the Wellcome Trust Four-Year PhD Programme in Integrative Neuroscience at Cardiff University, UK. Here, Tom discovered new mechanisms controlling neural circuit formation in the mammalian CNS and PNS. Subsequently, at the MRC Protein Phosphorylation & Ubiquitylation Unit, he led several studies that revealed groundbreaking insights into the regulation of physiological mitophagy across multiple organ systems, with significant relevance for neural integrity. In 2018, he was recruited to the University of Helsinki and appointed Tenure-Track Assistant Professor of Mitochodrial Medicine. In 2021, Tom was elected a Scholar of the FENS-Kavli Network of Excellence. The McWilliams lab deciphers the metabolic and mechanistic regulation of autophagy in tissue development, degeneration, and repair.

POSTERS

Posters will be presented on Slack platform on both days. The presentations have been divided more or less in two, with always a half of the presenters available on one day; these days are listed below. If the presenter is available on both days, they have implicated so on their poster channel.

Here you can find the list of poster presenters, their poster/channel number and the day of presentation. The provided titles and abstracts can be found on the following pages.

Poster No	Presenter name	Institution	Presentation day
1	Adrien Gigliotta	University of Helsinki	Thursday
2	Amirreza Asayesh	University of Helsinki	Friday
3	Cecilia Cannarozzo	University of Helsinki	Friday
4	Marta Saez Garcia	Neuroscience Center	Friday
5	Nikhil Singh	Yenepoya Research Centre	Thursday
6	Milo Grotell	University of Helsinki	Thursday
7	Saeed Montazeri Moghadam	University of Helsinki	Friday
8	Pushpa Khanal	University of Helsinki/Minerva	Thursday
9	Alessia Gallucci	University of Milano-Bicocca	Thursday
10	Emmi Pentikäinen	University of Helsinki	Friday
11	Ying Chieh Wu	University of Helsinki	Thursday
12	Anna Ptukha	University of Helsinki	Friday
13	Julius Rönkkö	University of Helsinki	Thursday
14	Jussi Tiihonen	University of Helsinki	Thursday
15	Ceren Pajanoja	University of Helsinki	Friday
16	Pilar Cerveró	Universidad de Salamanca	Friday
17	Raquel Flores	INCYL, USAL	Thursday
18	Andrea Álvarez Vázquez	University of Salamanca	Thursday
19	Adriana Della Pietra	University of Eastern Finland	Friday
20	Alex Subias Gusils	Autonomous University of Barcelona	Friday
21	Ilja Salakka	University of Helsinki	Thursday
22	Adam Alvarez	Autonomous University of Barcelona	Thursday
23	Daniel Garton	University of Helsinki	Friday
24	Tommi Aho	University of Helsinki	Friday
25	Viola Helaakoski	University of Helsinki	Thursday
26	Jonatan Panula	University of Helsinki	Friday
27	Carla Filipa Simões Henriques	Instituto Gulbenkian de Ciência	Thursday

POSTERS

1 Genetic background modulates the effect of chronic psychosocial stress on microglial function

Adrien Gigliotta, Federica Cafaro, Mikaela Laine , Kalevi Trontti and Iiris Hovatta

SleepWell Research Program and Department of Psychology and Logopedics, Faculty of Medicine; and Neuroscience Center, Helsinki Institute of Life Science HiLIFE, University of Helsinki, Finland

Anxiety disorders are the most prevalent psychiatric disorders and chronic psychosocial stress significantly contributes to their onset. Using the mouse chronic social defeat stress model (CSDS), our group recently identified myelin plasticity as a substantial response to stress, which varied across brain regions, and was genetically controlled. Critically, changes in myelin thickness varied between stress resilient (mice that did not develop social avoidance after CSDS) and susceptible (mice that developed social avoidance after CSDS) animals. Microglia are activated by chronic psychosocial stress and their activation and morphological modifications have been suggested to contribute to the development of anxiety-like states. We hypothesized that microglia-OPCs crosstalk is involved in the regulation of myelin plasticity and in resilience and susceptibility to psychosocial stress. Using an immunohistochemical approach in the anterior cingulate cortex, we compared the number of activated microglia (CD68+/IBA1+ cells) and the number of microglia engulfing OPCs (PDGFRa+ /IBA1+ cells) between C57BL/6NCrl (B6; innately non-anxious and mostly stress resilient) and DBA/2NCrl (D2; innately anxious and mostly stress susceptible) mice after CSDS. While CSDS did not affect the number of activated microglia, the area of CD68+ lysosomes was larger in B6 susceptible animals compared to controls, suggesting microglial activation after CSDS. We observed a smaller number of microglia engulfing OPCs and a longer distance between OPCs and the closest microglia in D2 susceptible animals compared to controls, indicating fewer microglia-OPC interactions. Our RNA-sequencing data from the medial prefrontal cortex after CSDS indicate that differentially expressed genes associated with microglia activation and phagocytosis are overrepresented among the differentially expressed genes in B6 susceptible vs control mice. Taken together, our results suggest that chronic psychosocial stress affects microglial function and that the effects are different in resilient and susceptible animals and in different mouse strains, highlighting the importance of considering the genetic background in the study of stress-induced activation of microglia.

POSTERS

2 Developing a disposable aEEG cap for NICU

Amirreza Asayesh, Sampsa Vanhatalo, Elina Ilen

Department of Neuroscience, Faulty of Medicine, University of Helsinki; and Department of Desgin, School of Arts, Design and Architecture, Aalto University

Background and Purpose: Long-term EEG monitoring is quite challenging with current EEG setups. Challenges Intensificate while aEEG recording from newborn infants at NICU as skin preparation by abrading or puncturing the stratum corneum and obstacles of multiple checkups of the skin-electrode interface for a stable signal quality in the long-term recordings. Materials and Methodology:A table of the requirements for an ideal cap, electrode, skin-electrode interface, and user experience was listed and described based on the needs for long-term aEEG recording from the newborn infants at NICU. We compared novel materials as conductive textiles (textured and woven), conductive velcro, sponge, super absorbent hydrogel (SAP), and thick-layer and thin-layer hydrofiber (HF) to conventional skin-electrode interfaces; as electro gel, tensive gel, conductive cream, and conductive paste to analyze each material's performance. Furthermore, comparing the connection stability between the AgCl electrode and the skin surface in the long-term recording. Then we compared the materials in a solution dehydration test to clarify the electrodes' ability to store conductive solutions for extended periods in open space and incubator mimicking space. The performance of chosen materials (Sponge, SAH, HF, and Sticker electrode) from the evaporation study compared on an armband sleeve for long-term biosignal recording on an unprepared skin. Then The performance of HF as the final material compared to a sticker electrode impregnated with tensive gel (to make it stable for long-time recordings) on a headband while recording biosignals from the forehead and impedance measurement. Results: According to the electrode requirements, conductive textiles had poor performance in absorbing and storing conductive solutions. Also, conductive velcros had rigid coated surfaces that could irritate the infant's skin. SAP and HF had similar outcomes in the Solution dehydration test, and they remained almost 75% after 6 hours and 20% hydration after 24 hours in an incubator mimicking environment. The sponge material was dehydrated completely before 12 hours of recording. The SAH material had a fragile structure and higher powerline (50hz) amplitude RMS component after 12 hours of recording. The electrical impedance for HF was significantly lower than tensive gel impregnated sticker electrode on an unprepared forehead skin, and the impedance remained under 15kΩ in 10Hz frequency for 18 hours after the recording started.

POSTERS

3 Ketamine reinstates visual plasticity in adult mice in a dose-independent manner

Cannarozzo C, Steinzeig A, Casarotto PC, Castrén E.

University of Helsinki,Neuroscience Center HILIFE, Helsinki, Finland

The ocular dominance plasticity (ODP) in the visual cortex is an established model to study activity-dependent plasticity: the forced closure of one eye (monocular deprivation) causes a shift in ocular dominance. Chronic treatment with fluoxetine, a classical antidepressant, has been shown to induce a state of juvenile-like plasticity in the adult mouse visual cortex, which was previously thought to be restricted only to a brief postnatal period. Since classical antidepressants take several weeks before their beneficial effects appear, research has been focusing on finding solutions to induce ODP in shorter latency. Ketamine has been described as a rapid-acting antidepressant in many preclinical and clinical trials, and the underlying mechanism is still under debate. Recently, we have reported that ketamine and its active metabolite 2R,6R-Hydroxynorketamine (R,R-HNK, both at 10 mg/kg) reinstate visual cortex plasticity in the adult mouse, with ketamine showing a weaker ocular dominance shift than R,R-HNK and fluoxetine. To assess whether this could be explained by the dose used, here we tested a higher or lower dose (20 or 3 mg/kg) of ketamine and the same dose of R,RHNK administered three times, every two days, during one week of monocular deprivation. Notably, these different doses resulted in comparable shifts in ocular dominance, yet still weaker than the ones obtained with R,R-HNK or fluoxetine, therefore suggesting a dose-independent mechanism for ketamine-induced ODP.

POSTERS

5 Development and Functional Characterisation of an in-vitro model of Ischemic Stroke

Nikhil Singh, Arnab Datta

Yenepoya Research Center, Mangalore, India

Background: Ischemic stroke is one of the major causes of death and disability worldwide with limited treatment options. There is an urgent need to develop effective and cheaper therapies. An in-vitro model is a useful tool for basic and pre-clinical research on ischemic stroke. These models are used to understand the cell-type-specific mechanisms and signalling. Objective: The primary objective was to establish hypoxia (HYP) and oxygen-glucose deprivation (OGD) model using cultured brain cells of neuronal and glial origin and subsequent estimation of the morphological and metabolic damage in a time-dependent manner. Methods: Cultured neuron (Neuro-2A, N2a) and astrocyte (CTX-TNA2) cells were exposed to variable durations of HYP and OGD spanning from 4 to 24 h to cover the acute and subacute phases of ischemic injury. A dissolved oxygen meter was used to confirm the persistent reduction of oxygen levels below 2% during OGD. Serum deprivation and retinoic acid treatment was used to induce differentiation of culture brain cells. Trypan blue and simple inverted microscopy were used to determine cell death and any change in cellular morphology. MTT assay was used to determine metabolic activity of the cells following HYP and OGD. Results: Forty-eight hr of serum deprivation was used to induce cellular differentiation. During early phases (4-8hr) of OGD and hypoxic injury, the relative metabolic activity of undifferentiated and differentiated neurons compared to control neurons was equal to or higher than the astrocytes. Variable changes in the metabolic activity of differentiated astrocytes were observed between 8hr and 16hr of hypoxic injury, while the metabolic activity of the neurons was reducing consistently with an increase in the duration of injury. Clearly, neurons and astrocytes responded differently to identical durations of hypoxic and ischemic stress. Conclusion: An in vitro OGD model has been established using cultured brain cells and validated using an MTT assay. This model can be used for mechanistic studies or for testing the cerebro-protective efficacy of novel chemical or biological entities.

POSTERS

6 Conditioned Reward of Opioids is Impaired in GABA-A Receptor δ Subunit Knockout

Milo Grotell, Elena de Miguel, Juho Aaltio, Aino K. Manner, Mikko Vahermo, Jari Yli-Kauhaluoma, Anni-Maija Linden, Teemu Aitta-aho, Esa R. Korpi

Department of Pharmacology, Faculty of Medicine; and Drug Discovery Program, Division of Pharmaceutical Chemistry and Technology, Faculty of Pharmacy, University of Helsinki, Helsinki, Finland

Gamma-Aminobutyric acid (GABA) is the primary inhibitory neurotransmitter in the mammalian brain. In the brain, GABA affects multiple different GABA-receptors. These receptors can be divided into ionotropic and metabotropic classes. The ionotropic effects are mainly mediated via A-type GABA receptors (GABAARs). Furthermore, GABAARs are mainly expressed in the synapses. However, there are also extrasynaptically expressed subpopulations of those receptors, which often contain delta-subunit.Interestingly delta-subunit GABAARs (d-GABAARs) are expressed in multiple locations needed for reward and populated with opioid receptors. Due to the lack of a specific d-GABAAR antagonist, a transgenic mouse model genetically lacking the d-GABAARs is used instead (d-KO). As opioids, like morphine, are known to be highly rewarding and are known to produce addiction, we wanted to evaluate if d-GABAAR-modulation could play a role in reward production. We used the conditioned place preference paradigm to evaluate if the morphine-induced reward is altered in d-KO mice compared to their littermates. We observed that d-KO mice did not produce conditioned place preference, whereas their littermates did. Our finding suggests that d-GABAAR-mediated modulation is crucial for normal reward. The underlining mechanism remains debatable, but it could be that d-GABAAR-mediated autoinhibition counteracts opioid-produced inhibition of GABAergic interneurons.

POSTERS

7 Building an Open Source Classifier for the Neonatal EEG Background

Moghadam, S.M. (1), Pinchefsky, E. (2), Tse, I. (1), Marchi, V. (1,3), Kohonen, J. (4), Kauppila, M. (1), Airaksinen, M. (1,5), Tapani, K. (1), Nevalainen, P. (1), Hahn, C. (6), Tam, E.W. (6), Stevenson, N.J. (7), and Vanhatalo, S. (1,8).

1 BABA Center, Pediatric Research Centre, Department of Clinical Neurophysiology, Children's Hospital and HUS Diagnostic Center, Helsinki University Hospital and University of Helsinki, Helsinki, Finland,
2 Division of Neurology, Department of Paediatrics, Sainte-Justine University Hospital Centre, University of Montreal, Montreal, QC, Canada,
3 Department of Developmental Neuroscience, Stella Maris Scientific Institute, IRCCS Fondazione Stella Maris Foundation, Pisa, Italy,
4 Department of Computer Science, Aalto University, Espoo, Finland,
5 Department of Signal Processing and Acoustics, Aalto University, Espoo, Finland,
6 Department of Paediatrics (Neurology), The Hospital for Sick Children and University of Toronto, Toronto, ON, Canada,
7 Brain Modelling Group, QIMR Berghofer Medical Research Institute, Brisbane, QLD, Australia,
8 Neuroscience Center, Helsinki Institute of Life Science, University of Helsinki, Helsinki, Finland

Neonatal brain monitoring in the neonatal intensive care units (NICU) requires a continuous review of the spontaneous cortical activity, i.e., the electroencephalograph (EEG) background activity. This needs development of bedside methods for an automated assessment of the EEG background activity. We present development of the key components of a neonatal EEG background classifier, starting from the visual background scoring to classifier design, and finally to possible bedside visualization of the classifier results. A dataset with 13,200 5-minute EEG epochs (8–16 channels) from 27 infants with birth asphyxia was used for classifier training after scoring by two independent experts. We tested three classifier designs based on 98 computational features, and their performance was assessed with respect to scoring system, pre- and post-processing of labels and outputs, choice of channels, and visualization in monitor displays. The optimal solution achieved an overall classification accuracy of 97% with a range across subjects of 81–100%. Our results showed that an automated bedside classifier of EEG background is achievable.

POSTERS

8 GAS7 initiates new dendritic spines in activation-dependent manner

Pushpa Khanal (1,2), Zoran Boskovic (3), Aruna Ghimire (1), Patricio Opazo (3), Pirta Hotulainen (1)

1 Minerva Foundation Institute for Medical Research, Helsinki, Finland
2 HiLIFE-Neuroscience Center, University of Helsinki, Helsinki, Finland
3 Clem Jones Centre for Ageing Dementia Research, The Queensland Brain Institute, The University of Queensland, Brisbane, Australia

Dendritic spines are small membrane-protrusions along neuronal dendrites and are the major postsynaptic target of excitatory synapses. They are highly dynamic structures whose shape, size, and number change throughout development as well as in response to the pattern and strength of synaptic activity. The actin cytoskeleton is known to have an important role in the morphogenesis and structural remodeling of dendritic spines. However, the exact mechanism of dendritic spine initiation is largely unknown. In this study we tested a selection of BAR domain containing proteins and identified GAS7 as a novel dendritic spine initiation factor. Overexpression of GAS7 in rat hippocampal neuronal cultures increased the dendritic spine density whereas the shRNA knock-down of GAS7 significantly decreased the spine density. Studies in rat hippocampal neuronal cultures and organotypic hippocampal slices indicate that GAS7 is localized to dendritic spines as well as to patches on the dendritic shaft plasma membrane. Live cell time-lapse imaging showed that the localization of these patches correlates with new spine formation. Moreover, treatment of organotypic hippocampal slices with bicuculline (GABAA receptors antagonist) leads to enrichment of the localization of GAS7 to the dendritic shaft patches. These results suggest that GAS7 is a novel dendritic spine initiation factor that can induce the formation of new spines in an activity-dependent manner.

POSTERS

9 Combining tDCS and CRT for the treatment of eating disorders patients

Gallucci, A.(1,2), Bergamelli, E. (3), Bertelli, S. (4), D'Agostino, A. (3,4), Mattavelli, G. (5), Gambini O. (3,4,6), Romero Lauro, L.J. (2,7)

1 Ph.D. program in Neuroscience, School of Medicine and Surgery, University of Milano-Bicocca, Monza, Italy.
2 NeuroMi (Neuroscience Center), University of Milano-Bicocca, Italy
3 Department of Health Sciences, University of Milan, Milan, Italy
4 Department of Mental Health, ASST Santi Paolo e Carlo, Milan, Italy
5 School of Advanced Studies, IUSS, Pavia, Italy
6 CRC "Aldo Ravelli" for Neurotechnology and Experimental Brain Therapeutics, University of Milan Medical School, Milan, Italy 7Department of Psychology, University of Milano-Bicocca, Milan, Italy

Objective. Cognitive Remediation Therapy (CRT) significantly improves set-shifting and central coherence in patients suffering from Anorexia Nervosa (AN) or Bulimia Nervosa (BN). Evidence suggested that the left dorsolateral prefrontal cortex (lDLPFC) might be crucially involved in the neurobiological mechanisms underlying ED's cognitive symptoms. Thus, combining CRT with the Transcranial Direct Current Stimulation (tDCS), despite still unexplored, could represent a promising method to boost therapeutic interventions. Method. Seven patients with AN and five with BN were randomly assigned to receive anodal or sham tDCS over the lDLPFC while performing 3 weeks- CRT intervention. Neuropsychological and clinical evaluations were performed to assess patients' cognitive skills and core symptoms at baseline and immediately after the combined treatment. Results. Preliminary results showed that the drive for thinness, personal alienation, interpersonal difficulties, affective problems as well as the global psychological maladjustment subscales of the Eating Disorder Inventory-3 (EDI-3) significantly decreased at the post-treatment evaluation in both the whole sample and in the anodal subgroup, while no effects were observed in the sham group. Conclusion. Our exploratory data suggest that the combined treatment modulated some of the clinical symptoms of patients, despite the EDI-3 results lack of consistency with the other clinical and neuropsychological tests.

POSTERS

10 Benefits of choir singing on complex auditory encoding in older adults

Emmi Pentikäinen (1), Lilli Kimppa (1), Tommi Makkonen (1), Anni Pitkäniemi (1), Ilja Salakka (1), Petri Paavilainen (1), Mari Tervaniemi (1,2), Teppo Särkämö (1)

1 Cognitive Brain Research Unit, Department of Psychology and Logopedics, Faculty of Medicine, University of Helsinki, Helsinki, Finland;
2 CICERO Learning Network, University of Helsinki, Helsinki, Finland

Normal aging is accompanied by difficulties in hearing and auditory information processing, especially in more complex sound environments. Choir singing requires fast and efficient processing of multiple sound features and could therefore potentially mitigate the deleterious effects of aging on complex auditory encoding, but this has not been studied previously. Using electroencephalography measurements in healthy older adult (age ≥ 60 years) choir singers and control subjects, we recorded auditory event-related potentials (ERPs) in two simple oddball conditions, in which the pitch and spatial location of the sounds was varied, as well as in a complex oddball condition, which involved higher-level encoding of abstract regularities in both pitch and location information, based on an implicit rule combining these two features. We analyzed standard P1 and N1 responses and change-related mismatch negativity (MMN) responses in left, middle, and right fronto-central regions of interest (ROIs) in each condition. In the simple pitch and location conditions, the choir singers had smaller N1 responses across all ROIs compared to the control subjects, whereas the MMN responses did not differ between groups. In the complex condition, the choir singers showed a larger MMN than the controls specifically in the left ROI, which also correlated with better executive functioning measured with a verbal fluency test. These results suggest that regular choir singing is associated both with more e

POSTERS

11 iPCS-derived APPswe mutant pericytes produce beta-amyloid and induce the activatation of co-cultured astrocytes

Ying-Chieh Wu (1), Taisia Rolova (1), Šárka Lehtonen (1,2) and Jari Koistinaho (1,2)

1 Neuroscience Center; University of Helsinki; Helsinki; Finland.
2 A.I.Virtanen Institute for Molecular Sciences; University of Eastern Finland; Kuopio; Finland.

Cerebrovascular dysfunction has been reported in Alzheimer's disease (AD) brains, but its underlying mechanism remains unclear. Cerebral amyloid angiopathy (CAA) is a form of vascular dysfunction caused by beta-amyloid (Aβ) accumulation in brain vessel walls. CAA is present in nearly 90 percent of AD patients suggesting it could be the primary pathology for vascular dysfunction in AD. The blood vessels in the brain possess unique properties, termed blood-brain barrier (BBB), composed of endothelial cells, pericytes, and astrocyte endfeets. Pericytes wrapping around endothelial cells are crucial for vasculature stabilization. Substantial pericyte loss has been reported in AD correlating with the BBB breakdown. We wonder if pericytes play a role in AD-associated vascular dysfunction and CAA pathology. To address these questions, we generated iPSC-derived pericytes from patients harboring APP Swedish mutation (KM670/671NL) and investigated the underlying mechanisms.

POSTERS

12 Eye movements as a potential strategy for fine stereoacuity

Anna Ptukha (1,2,3)

1.Doctoral Programme Brain & Mind
2.Cognitive Brain Research Unit
3.High Performance Cognition group
University of Helsinki

Perceived positions of stationary objects in the three-dimensional (3D) space and distances between them are stable according to everyday experience. Meanwhile, eyes, head and body move constantly, and the two-dimensional (2D) projections of the visual scene on the retinas undergo ongoing changes. Even during one precise fixation, so-called fixational eye movements (FEMs) occur a few times per second. Time period between consecutive FEMs is enough to perceive a 3D scene, but several FEMs usually occur in one fixation. Why then the perceived scene does not change with FEMs during fixation? The problem of deriving 3D scene from several pairs of 2D projections is ambiguous considering humans do not have precise readout of their eye positions. The assumption about stability of spatial positions of objects potentially helps to reduce the ambiguity. Binocular vision creates representation of space from differences between images projected to the retinae of two eyes. Considering that fine stereo information is lost when differences between 2D projections change after FEM, how can humans achieve high stereoacuity under FEMs? In psychophysics and eye tracking experiment, we found that a common solution is assigned to pairs of 2D projections formed from a given 3D scene during possible FEMs. We found that humans can exhibit stereoacuity higher than half of typical FEM amplitude, thus only distinct patterns of FEMs should appear, allowing high stereoacuity along only distinct directions.

POSTERS

13 Mutant calcium regulator IP3R3 in ER-mitochondria contact sites causes hereditary neuropathy

Julius Rönkkö (1), Svetlana Molchanova (1,2), Yago Rodriguez (1), Mari Auranen (3), Jussi Toppila (4), Jouni Kvist (1), Anastasia Ludwig (5), Anders Paetau (6), Claudio Rivera (5,7), Henna Tyynismaa (1,5,8), Emil Ylikallio (1,3)

1 Stem Cells and Metabolism Research Program, University of Helsinki, Finland;
2 Molecular and Integrative Biosciences Research Program, University of Helsinki, Finland; 3 Clinical Neurosciences, University of Helsinki and Helsinki University Hospital, Finland;
4 Department of Clinical Neurophysiology, Medical Imaging Center, Helsinki University Central Hospital, Finland;
5 Neuroscience Center, Helsinki Institute of Life Science, University of Helsinki, Finland;
6 Department of Pathology, HUSLAB and University of Helsinki, Finland;
7 Institut de Neurobiologie de la Méditerranée INMED UMR901, Marseille, France;
8 Department of Medical and Clinical Genetics, University of Helsinki, Finland

Background: Charcot-Marie-Tooth neuropathy (CMT) is a heterogeneous disorder that affects 1:2500 people. ITPR3 encodes type 3 inositol 1,4,5-trisphosphate receptor (IP3R3), one of three such receptors in humans together with IP3R1 and IP3R2. IP3R3 is localized to ER-mitochondria contact sites where it facilitates movement of Ca^{2+} between the organelles. Here we studied the causative role of ITPR3 variants in patients with CMT and the role of IP3 receptors in stem cells.
Methods: We used whole exome sequencing to identify disease-causing variants in four affected individuals with CMT in an autosomal dominant family. Skin fibroblasts from two affected individuals were analyzed by Western blotting, quantitative reverse transcription PCR and Ca^{2++} imaging. Knockouts of IP3 receptors in induced pluripotent stem cells (iPSC) were generated with CRISPR-Cas9.
Results: Affected individuals had onset of symmetrical neuropathy with demyelinating features at around age 30, showing signs of gradual progression with severe distal leg weakness and hand involvement in the proband at age 64. Exome sequencing identified a heterozygous ITPR3 p.Val615Met variant segregating with the disease. Altered Ca^{2+} transients in p.Val615Met patient fibroblasts suggested that the variant has a dominant negative
effect on IP3R3 function. For further analysis of IP3 receptor function, we generated iPSC-knockouts of the three receptors.
Conclusions: With the genetic and functional evidence of our study, we conclude that ITPR3 is a new disease gene for Charcot-Marie-Tooth disease. Our results suggest that abnormal calcium signaling contributes to degeneration of peripheral nerves. IP3R knockout iPSC cell models provide a tool for further characterization of the receptors.

POSTERS

14 Can a human see a single photon?

Tiihonen, J. (1), Dovzhik, K. (2), Tavala, A. (3), Zeilinger, A. (3), Ala-Laurila, P. (1,2)

1 Molecular and Integrative Biosciences Research Programme, University of Helsinki, Helsinki, Finland
2 Department of Neuroscience and Biomedical Engineering, Aalto University, Espoo, Finland
3 Institute for Quantum Optics and Quantum Information - Vienna, Austrian Academy of Sciences, Vienna, Austria

The human visual system is surprisingly sensitive to dim light stimuli. Classical studies in the 20th century quantified this notion by showing that we can reliably detect as few as 5 to 10 absorbed photons. This result consequently lead to the question whether humans could see just a single photon. However, the inherent photon variability of conventional light sources restricted the possibility to answer this question. Now, recent advances in quantum optics have re-ignited keen interest in this question, as newly introduced single-photon sources have reduced photon variability when compared to conventional light sources. Nonetheless, a prerequisite for using these single-photon sources is that the reduced variability can also be seen in biological responses. However, this prerequisite has not been tested in a rigorous way. Here, we now show for the first time that this prerequisite fails due to the losses of single-photon signals in the retina. Consequently, the reduced variability in biological responses can not be detected on a biologically relevant time scale and humans can basically never see a single photon.

POSTERS

15 Decline of Broad Ectodermal Pluripotency Leads to Neural Crest Formation

Ceren Pajanoja (1,2),Laura Kerosuo (1,2)

1 Department of Biochemistry and Developmental Biology, Faculty of Medicine, Medicum, University of Helsinki, Helsinki, Finland
2 Neural Crest Development and Disease Unit, National Institute of Dental and Craniofacial Research, National Institutes of Health, Bethesda, USA

Neural crest (NC) is an embryonic stem cell population that forms the craniofacial skeleton, peripheral nervous system and cells of the hormonal regulatory system. Despite its ectodermal origin, some NC derivatives show features of mesodermal or endodermally derived cells, which has puzzled researchers for decades and suggests the NC has an exceptionally high stem cell potential. The molecular mechanisms of how this stemness is acquired during early ectodermal development are not known.To uncover the process we performed Multiplex Spatial Transcriptomics (MST) at single cell level on chick embryo midbrain samples from multiple developmental stages. Surprisingly, our results show that the entire ectoderm at the neural fold stage, thus long after gastrulation, contains many undecided cells that could still become any of the ectodermal cell types (NC, central nervous system or epidermis), and which co-express the pluripotency factors. This contradicts previous assumptions of fully committed ectodermal domains at this stage. By the neural tube closure stage the NC domain has the highest expression of Nanog, which we think is linked to the broad stem cell potential maintained in the NC.Additionally, our bulk RNAseq from 12 consecutive stages from gastrula to NC migration show a consistent maintenance of pluripotency in the NC. Combined, we propose NC gains its high stemness by a process of gradual rather than abrupt decline of pluripotency in the ectoderm post gastrulation.

POSTERS

16 Effect of the Src inhibitory connexin43 region on lung cancer stem cells.

Cerveró-García, P., García-Vicente,L., Tabernero, A.

Universidad de Salamanca, Departamento de Bioquímica y Biología Molecular- Instituto de Neurociencias de Castilla y León INCyL, Salamanca, Spain; and
Instituto de Investigación Biomédica de Salamanca IBSAL, Área de Neurociencias, Salamanca, Spain

Connexin43 (Cx43) is a gap junction forming protein. Multiple studies have shown that Cx43 is related to cancer pathogenesis. Cx43 can suppress cancer cell growth through a mechanism mediated by its intracellular C terminus. Previous results indicate that Cx43 performs anti-tumourigenic effects in glioma cells by inhibiting the activity of c-Src oncoprotein. Based on this mechanism, a cell penetrating peptide was designed (Tat-Cx43266-283), which preserves the capacity of inhibiting Src on different in vitro and in vivo models of glioma. Src is a relevant oncogene, involved in numerous signaling pathways related to different cancer types, including lung cancer and lung cancer brain metastasis. Consequently, we asked whether the designed Src inhibitory peptide could impair brain metastasis as shown in primary brain tumours. To do so, we first analysed the effect on lung cancer stem cells. Using the human lung cancer cell line A549, we obtained a stem cell subpopulation from differentiated A549 cells. We carried out cell survival and protein expression assays at different times after the treatment with Tat-Cx43266-283 in differentiated and stem subpopulations of this cell line. We show the effects of Tat-Cx43266-283 on the expression of Cx43 and other Cx isoforms, on stem cell markers and on Src activity.These results aim us to explore the effect of the Src inhibitory peptide on lung brain metastasis models to address Tat-Cx43266-283 effects within the brain microenvironment.

POSTERS

17 Effect of TAT-Cx43266-283 on the expression and localization of Connexin43

Flores-Hernández, R., Talaverón, R. García-Vicente, L., Álvarez-Vázquez, A., Medina, J.M., Tabernero, A.

Departamento de Bioquímica y Biología Molecular, Instituto de Neurociencias de Castilla y León (INCYL), Universidad de Salamanca, Salamanca, Spain Instituto de Investigación Biomédica de Salamanca (IBSAL), Salamanca, Spain

Gliomas are the most common and aggressive primary brain tumors. Glioblastoma, its most malignant grade, are composed by a heterogeneous population of cells, including some called glioma stem cells (GSCs). These cells are highly tumorigenic and resistant to standard therapies. Connexin43 (Cx43) is an integral membrane protein, which in the brain is mainly found forming gap junctions in astrocytes. In addition, Cx43 forms hemichannels that release paracrine signals, and possesses channel-independent functions, including intracellular interaction with signalling molecules, such as the oncoprotein c-Src. Importantly, Cx43 inhibits c-Src activity by recruiting c-Src and its inhibitors. Based on this property, we developed a cell-penetrating peptide containing the region of Cx43 that interacts with c-Src (TAT-Cx43266-283). TAT-Cx43266-283 inhibits c-Src activity, reducing the growth and invasion of glioma cells without affecting neurons and astrocytes in several preclinical glioma models. In this study we addressed the effect of TAT-Cx43266-283 on Cx43 in glioma cells and the tumor microenvironment in a murine glioma model. To do so, Gl261-GSCs were intracranially injected in C57BL/6 mice. For the treated group, TAT-Cx43266-28 was coinjected with Gl261-GSCs and IP administered twice a week. We first analyzed different immunohistochemical methods. Then, we studied the effect of TAT-Cx43266-283 on the expression and localization of Cx43 in glioma cells and astrocytes.

POSTERS

18 Effect of the peptide TAT-Cx43266-283 on neural stem cells with EGFR alteration

Álvarez-Vázquez, A. (1), Segura-Collar, B. (2), Sánchez-Gómez, P. (2), Tabernero, A. (1)

1 Instituto de Neurociencias de Castilla y León (INCyL), University of Salamanca, Salamanca, Spain
2 3 Neuro-oncology Unit, Instituto de Salud Carlos III-UFIEC, Madrid, Spain

Glioblastomas are one of the most malignant tumours worldwide. Among the causes of such malignancy is a subpopulation of tumour cells with stem cell properties known as Glioma Stem Cells (GSCs). These cells are resistant to standard treatments, such as temozolomide. Several studies have proposed Neural Stem Cells (NSCs) from the subventricular zone (SVZ) as a possible origin for GSCs. The transition of NSCs to GSCs frequently concurs with epidermal growth factor receptor (EGFR) alterations. Our group designed a cell penetrating peptide based on connexin43 (TAT-Cx43 266-283) that inhibits the activity of the oncoprotein c-Src and therefore targets GSCs, increasing survival rates in glioma-bearing mice. Because Src is involved in EGFR signaling, we explored the effect of TAT-Cx43 266-283 in the transition of NSCs to GSCs. For this purpose, we analysed the cell growth of SVZ NSCs (Control-NSCs), NSCs with EGFR amplification (EGFRwt-NSCs) and NSCs with the mutant EGFRvIII (EGFRvIII-NSCs). Our results show that TAT-Cx43 266-283 specifically inhibited the growth of EGFRwt-NSCs and EGFRvIII-NSCs, without significant effects in Control-NSCs. Importantly, we found that temozolomide and other control peptides did not affect the cell growth of any of these NSCs. EGFR activity was analyzed by Western blot and our preliminary results show a reduction in the activity of EGFR, EGFRvIII and Src. These results stress the relevance of TAT-Cx43 266-283 as a future therapy against glioblastoma.

POSTERS

19 Molecular migraine treatment via inhibition of endoCBs-hydrolyzing enzymes

Della Pietra A., Giniatullin R., Savinainen J.R.

A. I. Virtanen Institute for Molecular Sciences, University of Eastern Finland, Kuopio, Finland; and
Laboratory of Neurobiology, Kazan Federal University, Kazan, 420008, Russia; and
Institute of Biomedicine, University of Eastern Finland, Kuopio, Finland

In migraine pain, cannabis has a promising analgesic action which, however, is associated with side psychotropic effects. To overcome these adverse effects of exogenous cannabinoids, we propose migraine pain relief via activation of the endogenous cannabinoid system (ECS) by inhibiting enzymes degrading endocannabinoids. To provide a functional platform for such purpose in the peripheral and central parts of the rat nociceptive system relevant to migraine, we measured by activity-based protein profiling (ABPP) the activity of the main endocannabinoid-hydrolases, monoacylglycerol lipase (MAGL) and fatty acid amide hydrolase (FAAH). We found that in trigeminal ganglia, the MAGL activity was 9-fold higher than that of FAAH. MAGL activity exceeded FAAH activity also in DRG, spinal cord and brainstem. However, activities of MAGL and FAAH were comparably high in cerebellum and cerebral cortex implicated in migraine aura. MAGL and FAAH activities were identified and blocked by the selective and potent inhibitors JJKK-048/KML29 and JZP327A, respectively. The high MAGL activity in trigeminal ganglia implicated in generation of nociceptive signals suggest this part of ECS as a priority target for blocking peripheral mechanisms of migraine pain. In the CNS, both MAGL and FAAH represent potential targets for attenuation of migraine-related enhanced cortical excitability and pain transmission.

POSTERS

20 Behavioural and metabolic effects of a restricted cafeteria diet in obese rats

Alex Subias-Gusils (1,2); Adam Álvarez (1,3); Noemí Boqué (4); Antoni Caimari (4,5); Josep Maria Del Bas (4); Montserrat Solanas (1,3); Rosa Maria Escorihuela (1,2)

1 Institut de Neurociències, Universitat Autònoma de Barcelona, Bellaterra, Spain
2 Departament de Psiquiatria I Medicina Legal, Facultat de Medicina, Universitat Autònoma de Barcelona, Bellaterra, Spain
3 Department of Cell Biology, Physiology and Immunology, Medicine School, Universitat Autònoma de Barcelona, Bellaterra, Spain
4 Eurecat, Centre Tecnologic de Catalunya, Technological Unit of Nutrition and Health, Reus, Spain
5 Eurecat, Centre Tecnològic de Catalunya, Biotechnology Area and Technological Unit of Nutrition and Health, Reus, Spain

Diet-induced obesity models are widely used to investigate dietary interventions for treating obesity. The current study was aimed to test the outcomes of a calorie cafeteria-restricted diet (CAF-R) and the supplementation with a polyphenolic compound (Oleuropein, -OLE-). Male rats were fed standard chow (STD) or cafeteria (CAF) diet for 8 weeks to induce obesity. Afterwards, the CAF group was divided into three groups: CAF, fed the same CAF diet; CAF-R, fed a restricted cafeteria diet; CAF-RO, fed restricted cafeteria diet supplemented with OLE (25 mg/kg*day). Biometric, food consumption, and serum parameters were measured. The two-bottle preference and taste reactivity tests were performed to evaluate sweet preference and hedonic responses to sucrose solutions. As expected, CAF diet significantly increased body weight and metabolic parameters associated with obesity . Interestingly, CAF-R diet attenuated the levels of obesity-related parameters. OLE did not seem to exert additional effects on biometric or metabolic parameters, although diminished the preference and the number of hedonic responses for the high sucrose concentrations compared with the other groups. These results indicate that CAF-R diet may be an efficient diet-based strategy to lose weight and restore obesity-associated metabolic alterations. In addition, CAF-RO diet seems to prevent high sweet solutions consumption.

POSTERS

21 Relationships between musical features and music-evoked emotions and memories

Salakka, I (1), Pitkäniemi, A (1), Pentikäinen, E (1), Mikkonen, K (2), Saari, P (3), Toiviainen, P (3), Särkämö, T (1)

1 University of Helsinki
2 Sentina Ltd
3 University of Jyväskylä

Music has the capacity to evoke both strong emotions and vivid personal memories. Previous studies have shown that the emotional experience of music is influenced by a combination of musical features. Here, our aim was to explore the relationship between music-evoked emotions and memories and how musical features can predict them. 113 older adults (age≥60 years) participated in a listening task in which they rated a total of 140 song excerpts (folk songs and songs from 1950s–1980s) on valence, arousal, emotional intensity, familiarity, and autobiographical salience. A set of 24 musical features were extracted from the songs using computational methods. PCA was applied resulting in six musical components, which were then used to predict the ratings in multiple regression analyses. All correlations between the ratings were positive and ranged from moderate to high (r=0.46–0.92). Emotional intensity had the highest correlation to both autobiographical salience and familiarity. Three musical components measuring pulse strength, brightness, and fluctuation in the low middle frequencies (200–800Hz) predicted both music-evoked emotions and memories. Emotional intensity (and valence to a lesser extent) mediated the predictive effect of the musical components on music-evoked memories. The results suggest that music-evoked emotions are strongly related to music-evoked memories in older adults and that music-evoked emotions and memories are predicted by the same core musical features.

POSTERS

22 Effects of cafeteria diet, restrictive feeding and exercise on rats sweet taste

Alvarez A (1,3), Subias-Gusils A (2,3), Munar F (2,3), Ordeix C (2,3), Escorihuela RM (2,3), Solanas M (1,3)

1 Dpt. Cell Biology, Physiology and immunology, Medicine School, Universitat Autònoma de Barcelona; and
2 Dpt. Psychiatry and Forensic Medicine, Medicine School, Universitat Autònoma de Barcelona; and
3 Neuroscience Institute, Universitat Autònoma de Barcelona, Bellaterra (Barcelona), Spain

It is well known that obesity presents sex differences, but differential effects on certain parameters such as the sweet test remain to be studied. We aimed to study how a previously characterized cafeteria restrictive diet (CAF-R), alone or in combination with exercise, affects the sweet taste and biometrical parameters in obese male and female rats. After inducing obesity with CAF feeding, animals were placed under a treadmill exercise (E) program and/or the CAF-R dietary intervention for 8 weeks. Then a sucrose preference test and a brief-access licking test with sucrose were performed. Male and female rats were studied in separate experiments, both using a 3X2 experimental design, and are not compared directly. Results showed a decrease of BW and BMI by CAFR diet in females, whereas in males this effect was only seen in BMI in exercised CAF-R animals compared with their control. Regarding the preference test, both sexes decreased sucrose intake when fed CAF diet, with exercise also decreasing intake in CAF fed animals. CAFR feeding increased sucrose intake and lick response in the brief-access test in both sexes. These results suggest CAF feeding might induce an anhedonic state that is partially reverted by CAFR feeding in both sexes. The treadmill intervention affected the sweet taste in both sexes and to a greater degree in females.

POSTERS

23 Endogenous GDNF upregulation increases brain serotonin system function

Garton, D.R. (1); Casserly, L. (1); Kopra, J.J. (2); Mätlik, K. (1); Olfat, S. (1,3); Piepponen, T.P. (2); Andressoo, J-O. (1,3)

1 Department of Pharmacology, Faculty of Medicine, Neuroscience Center & Helsinki Institute of Life Science, University of Helsinki, Helsinki, Finland.
2 Division of Pharmacology and Pharmacotherapy, Faculty of Pharmacy, University of Helsinki, Helsinki, Finland.
3 Division of Neurogeriatrics, Department of Neurobiology, Care Sciences and Society (NVS), Karolinska Institutet; Stockholm, Sweden.

The glial cell line-derived neurotrophic factor (GDNF) is an important nerve growth factor for the development and maintenance of a variety of cell types (Ibañez & Andressoo 2016). Since GDNF was first discovered as a potent neurotrophic factor for the growth and survival of dopamine neurons, its effects were hypothesized to be relatively dopamine-specific with no significant effects observed on serotonin (5-HT) (Lin et al., 1993; Lin et al., 1994; Hoffer et al., 1994). While the role and therapeutic potential of GDNF on the dopamine system is well established, knowledge on how GDNF affects the 5-HT system is sparse (Popova et al., 2016). To further investigate the potential role of GDNF on the 5-HT system, here we use genetic tools developed by our lab to both knock-out (Kopra et al., 2015) and upregulate (Kumar et al., 2016) GDNF from its endogenous locus. We show that while knocking out GDNF does not affect overall 5-HT levels in any observed area of the brain, GDNF hypermorphic mice have significantly upregulated 5-HT levels, release, reuptake, and behavioral response to the selective serotonin reuptake inhibitor fluoxetine. We further show that both developmental brain-specific GDNF and adult-onset striatal GDNF upregulation increases 5-HT. Overall, this study demonstrates that endogenous GDNF, while not necessary, is sufficient to induce changes to the 5-HT system, providing much-needed insight into the molecular mechanisms governing 5-HT neurobiology.

24 Temperament an quality of life in breast cancer survivors with chronic pain

Aho, T. (1), Lipsanen, J. (2), Kalso, E. (1), Harno, H. (1,3) & Sipilä, R. (1)

1 Division of Pain Medicine, Department of Anesthesiology, Intensive Care and Pain Medicine, University of Helsinki and Helsinki University Hospital, Finland
2 Department of Psychology and Logopedics, Faculty of Medicine, University of Helsinki, Helsinki, Finland.
3 Clinical Neurosciences, Neurology, University of Helsinki and Helsinki University Hospital, Finland

Background: Pain after breast cancer (BC) treatments have a negative effect on patient's health related quality of life (HrQoL). Psychobiological temperament influence on one's long-term adjustment of chronic pain via emotional reactivity, coping and health behavior. The Harm Avoidance (HA) temperament has been associated with greater burden of chronic disease in chronic pain patients. We aimed to study if pain-related and psychological factors mediate the effect of HA on HrQoL. Methods: We studied 273 BC treated patients who reported chronic pain at any site of the body. HrQoL and HA temperament were assessed by using the Short-Form-36 (SF-36) questionnaire and the Temperament and Character Inventory (TCI). Pain interference, pain catastrophizing, and anxiety and depressive symptoms were assessed. We used parallel mediation modelling, by using the PROCESS function V.2.16.1 in SPSS. Results: The total effect of HA on physical (β=-0.665, p<0,001) and mental (β=-1.071, p<0,001) HrQoL was significant. The effect of HA was fully mediated through depressive symptoms, pain catastrophizing, and pain interference on physical HrQoL and through depressive symptoms and anxiety for mental HrQoL. Conclusions: Psychological and pain-related variables mediate the effect of HA on impaired HrQoL in BC treated chronic pain patients. HA is a potential risk factor for psychological burden of chronic pain.

POSTERS

25 Sleep problems are associated with increased alcohol consumption

Viola Helaakoski (1), Jaakko Kaprio (1,2,3), Hanna M. Ollila (1,4,5,6), Antti Latvala (7,8)

1 Institute for Molecular Medicine, HiLIFE, University of Helsinki, Helsinki, Finland
2 Department of Public Health, University of Helsinki, Helsinki, Finland
3 National Institute for Health and Welfare, Helsinki, Finland
4 Broad Institute of MIT and Harvard, Cambridge, Massachusetts, USA
5 Center for Genomic Medicine, Massachusetts General Hospital, Boston, USA
6 Anesthesia, Critical Care, and Pain Medicine, Massachusetts General Hospital, Harvard Medical School, Boston, USA
7 Institute of Criminology and Legal Policy, University of Helsinki, Helsinki, Finland
8 Department of Medical Epidemiology and Biostatistics, Karolinska Institutet, Stockholm, Sweden

Alcohol is one of the most common psychoactive substances, and although legally available, it is associated with several health issues, including sleep problems. Despite the fact that acute effects of alcohol intake on sleep have been widely investigated, its longitudinal effects remain relatively underexplored. The objective of our research is to shed light on cross-sectional and longitudinal associations between alcohol consumption and sleep problems over time, and to elucidate the role of genetics in such associations. Using data from the Older Finnish Twin Cohort, we examined how short sleep duration and poor sleep quality correlate with heavy alcohol consumption and binge drinking during the period of 36 years. Cross-sectional regression analyses revealed there to be significant associations between sleep and alcohol traits at different time points, suggesting that increased alcohol intake correlates with decreased sleep quality and sleep duration over the years. Cross-lagged analyses indicate that both heavy drinking and binge drinking increase poor sleep quality over time, but not vice versa. The results from within-pair analyses showed too weak associations to draw any conclusions about genetic influences. In conclusion, our findings support previous literature in that short sleep duration and poor sleep quality are associated with increased alcohol use. Additionally, our results suggest that heavy and binge drinking increase sleep problems over time.

POSTERS

26 Functional changes in the salience network correlate with delusion intensity in first-episode psychosis patients

Jonatan Panula (1,2), Tuula Kieseppä (1,3), Jaana Suvisaari (4), Tuukka Raij (1,2)

1 Department of Psychiatry, Helsinki University and Helsinki University Hospital, Helsinki, Finland
2 Department of Neuroscience and Biomedical Engineering, and Advanced Magnetic Imaging Center, Aalto University School of Science, Espoo, Finland
3 Institute for Molecular Medicine Finland (FIMM), University of Helsinki, Helsinki, Finland
4 Mental Health Unit, National Institute for Health and Welfare, Helsinki, Finland

Reality distortion symptoms are characteristic of psychotic disorders, however, the brain correlates of delusions remain poorly known. To increase sensitivity, and validity for naturalistic brain functioning, we collected individual fMRI signal-time series, time-locked to movie stimulus, after remission of psychosis. These voxelwise signal time series were used to model corresponding fMRI signal time series during the first episode psychosis, characterized by delusions. From the Helsinki Early Psychosis Study (HEPS), we selected those patients who presented without delusions at the one-year follow-up. The magnitude of FEP-related functional brain alteration negatively correlated with the baseline delusion severity in bilateral insula. In addition, we observed a similar negative correlation in the anterior cingulate. Functional connectivity between both insulas with the precuneus was decreased in the baseline patient group when compared with control subjects and with the same patients at remission. The results support earlier evidence on involvement of the cortical hub regions in delusions, especially of the salience network (SN). Further studies should assess role of SN in (dys)regulation of meso-striatal dopaminergic pathways.

POSTERS

27 Functional specialization of social and asocial learnings

Simões-Henriques, C. (1), Marcos, J. (1), Sucena, E. (1), Vasconcelos, M.L. (2), Varela, S.A.M. (1,2), Oliveira, R.F. (1,2,3)

1 Instituto Gulbenkian de Ciência, Portugal
2 Champalimaud Centre for the Unknown, Portugal
3 ISPA-Instituto Universitário, Portugal

Social and asocial learnings are required to cope with the environment. They are behaviourally distinct. However, whether they use a single (general-purpose) or distinct (special-purpose) cognitive mechanisms remains elusive. To address this question, we firstly tested social and asocial learning abilities in 40 lines of the DGRP, a panel of several isogenic sequenced lines that together represent the genetic variation of a natural Drosophila melanogaster population. For asocial learning, individual flies had to learn to avoid an oviposition-site where they associated an odour (conditioned stimulus, CS) with a bitter taste (unconditioned stimulus, US). For social learning, flies had to learn the oviposition-site choice of a group of conspecifics previously exposed to the CS and the US. Secondly, we performed a Genome-Wide Association Study – a statistical correlation between the genetic variants in the DGRP and each learning phenotype. We obtained two completely different sets of candidate genes for social and asocial learnings. Thirdly, to functionally validate the role of each gene on each learning phenotype, we used GAL4/RNAi-UAS lines to knockdown the genes. We found genes that affect both learnings and also cases of specialization. This is the first experimental evidence supporting the special-purpose cognitive mechanism hypothesis. Future work should focus on the extent of this specialization, concerning the biochemical pathways and the neural circuitry