Final ENINET Meeting, May 19-23 2009 in Crete

Program

May 19th, Tuesday

20.00 Get-together for early arrivers in the lounge of the Fodele Beach Hotel

May 20th, Wednesday

Welcome note by Nektarios Tavernarakis

09.00 - 09.30 **Santiago Canals**, Institute de Neurosciencias de Alicante (CSIC-UMH)
FMRI of synaptic LTP reveals reorganization of network connectivity

09.30 - 10.00 **Huibert Mansfelder,** CNCR VU Amsterdam
Altered expression of protein networks involved in synaptic development, maintenance and function affect presynaptic plasticity in a mouse model of Frail X Syndrome

10.00 - 10.30 **Giovanni Marsicano**, INSERM Bordeaux
Bimodal control of food intake by the endocannabinoid system

10.30 - 11.00 *Coffee Break*

11.00 - 11.30 **Jack Mellor,** University of Bristol
Place cell firing patterns can induce long-term synaptic plasticity in the hippocampus

11.30 - 12.00 **Panayiota Poirazi**, IMBB-FORTH Crete
Modeling Memory Functions in single cells and small networks

12.00 - 12.30 **Matt Nolan**, University of Edinburgh
Tuning of synaptic responses in a cognitive circuit

12.30 - 13.00 **Detlev Schild**, University Göttingen
Gaining Information from the Time Domain of Ca^{2+}-Signals

13.00 - 15.00 *Lunch and Poster Session*

15.00 **General assembly**

Draft Agenda

- Welcome (Nektarios Tavernakaris)
- Development of the network (Stefan Eimer)
- Project administration (Joachim Bormann)
- Presentation of the new ENINET Website (Helene Marie)
- ESF proposal
- Discussion on the future of the network
- (cost-neutral extension of the project)

May 21st, Thursday

09.00 - 9.30 A**ttila Köfalvi** , CNC Coimbra
Presynaptic neuromodulation by cannabinoids in the dorsolateral striatum – new players besides CB1 and TRPV1 receptors

09.30 - 10.00 **Oliver Schlüter**, ENI Göttingen
The role of DLG-MAGUK signaling scaffolds in synaptic plasticity

10.00 - 10.30 **Anthony Holtmaat**, University of Geneva
Structural synaptic changes in cortical adaptive plasticity

10.30 - 11.00 *Coffee Break*

11.00 - 11.30 **Cécile Lebrand**, University of Lausanne
Transient neuronal populations are necessary for the formation of the corpus callosum

11.30 - 12.00 **Paola Pedarzani,** UC London
The slow afterhyperpolarising current in hippocampal neurons: a hub for neuromodulatory signals

12.00 - 12.30 **Dieter Chichung Lie**, Helmholtz Center Munich
Role of CREB in adult hippocampal neurogenesis

12.30 - 13.00 **Ola Hermanson,** KI Stockholm
Developmental neuroscience in the hiStone age

13.00 - 15.00 *Lunch and Poster Session*

15.00 - 15.30 **Valentina Emiliani**, Paris Descartes
Spatiotemporal control of neuronal activity by single and two photon holographic photoactivation patterns

15.30 - 16.00 **Bernard Lakowski**, Paris Pasteur
Spr-2 suppresses sel-12 presenilin mutations by a novel mechanism

16.00 - 16.30 **Pavla Jendelova**, Institute of Experimental Medicine Prague
Polymer scaffold seeded with stem cells – a tool for spinal cord injury repair

16.30 - 17.00 **Alberto Bacci**, EBRI Rome
Fast and slow self-inhibition of cortical neurons

17.00 - 18.00 *Coffee Break and Poster Session*

18.00 - 18.30 **Alberto Pascual**, University Seville
Role of GDNF during cerebral postnatal development and adulthood: Facts and hypotheses

18.30 - 19.00 **Oleg Shupliakov**, KI Stockholm
How synapses organize their periactive zone

19.00 - 19.30 **Yann Humeau**, INCI Strasbourg
Neuronal and synaptic physiology in mouse models of mental retardation

19.30 - 20.00 **Leszek Kaczmarek**, NENCKI Warsaw
Proteiolytic shaping of synapses

20.30 **Dinner Party**

List of speakers

1. **FMRI of synaptic LTP reveals reorganization of network connectivity**
   Santiago Canals, Cellular and Systems Neurobiology, Institute de Neurosciencias de Alicante (CSIC-UMH)

   I'd like to present my last findings on global networks plasticity triggered by LTP of synaptic transmission in the dentate gyrus. I've recently shown by combining fMRI, electrophysiological recordings and electric microstimulation in the rat, that local potentiation of synaptic strength within the hippocampus induces long-lasting changes in functional connectivity, including increased interhemispheric communication and recruitment of limbic networks.

2. **Altered expression of protein networks involved in synaptic development, maintenance and function affect presynaptic plasticity in a mouse model of Frail X Syndrome**
   Huibert Mansfelder, CNCR, VU University Amsterdam

3. **Bimodal control of food intake by the endocannabnoid system**
   Giovanni Marsicano, INSERM Bordeaux

4. **Place cell firing patterns can induce long-term synaptic plasticity in the hippocampus**
   Jack Mellor, University of Bristol

5. **Modeling Memory Functions in single cells and small networks**
   Panayiota Poirazi, IMBB-FORTH Crete

6. **Tuning of synaptic responses in a cognitive cirucuit**
   Matt Nolan, University of Edinburgh

7. **Presynaptic neuromodulation by cannabinoids in the dorsolateral striatum – new players besides CB1 and TRPV1 receptors**
   Attila Köfalvi, CNC Coimbra

8. **The role of DLG-MAGUK signaling scaffolds in synaptic plasticity**
   Oliver Schlüter, ENI Göttingen

9. **Structural synaptic changes in cortical adaptive plasticity**
   Anthony Holtmaat, Department of Basic Neuroscience CMU, University of Geneva

   To understand the synaptic, cellular and network mechanisms of circuit plasticity in relation to learning and memory, neurons need to be studied in the intact brain over extended periods of time. I will describe a procedure to image neurons and their synapses in the mouse neocortex, using long term high-resolution two-photon laser scanning microscopy through a chronic cranial window, followed by the ultrastructural reconstruction of imaged neurons, using serial section EM. Such studies have recently shown that proxies for synapses, such as dendritic spines and axonal boutons,

are dynamic structures, even in the adult brain. Whereas most spines are persistent for months, a small subset of dendritic spines can appear and disappear over days. This growth and retraction of spines, with synapse formation and elimination, is modulated by changes in experience.

10. **Transient neuronal populations are necessary for the formation of the corpus callosum**
Cécile Lebrand, Department of Cellular Biology and Morphology (DBCM), University of Lausanne, Switzerland

The corpus callosum (CC) is essential for numerous higher cortical functions. While its development has been shown to rely on midline glial cells, CC agenesis is associated with numerous human pathologies, suggesting that a range of developmental defects can result in abnormalities in this structure. Here, we show that two transient populations of midline neurons contribute to the formation of the CC. We report that these two populations of neurons enter the CC midline prior to the arrival of callosal pioneer axons. Using a combination of mutant analysis and in vitro assays, we demonstrate that CC neurons are necessary for normal callosal axon navigation. They exert an attractive activity on callosal axons, in part via Sema3C and its receptor Neuropilin-1. Our study reveals a novel and essential role for these neuronal populations in the pathfinding of a major cerebral commissure and raises new perspectives on pathophysiological mechanisms altering CC formation.

11. **The slow afterhyperpolarising current in hippocampal neurons: a hub for neuromodulatory signals**
Paola Pedarzani, UC London

12. **Role of CREB in adult hippocampal neurogenesis**
Dieter Chichung Lie, Helmholtz Center Munich

13. **Spatiotemporal control of neuronal activity by single and two photon holographic photoactivation patterns**
Valentina Emiliani, Neurophysiology and New Microscopy Laboratory, CNRS UMR 8154, INSERM U603, Université Paris Descartes, Paris France

Understanding how neurons process information by transducing synaptic inputs into action potentials is a fundamental problem in neuroscience. A promising approach to investigate this problem is to manipulate neuronal signals by photoactivation of caged compounds. However, this method has been severely constrained by limitations of pointing scanning illumination systems. To overcome this, we present a holographic microscope for single and two-photon (2P) photoactivation based on the combination of a spatial light modulator to control the patterning of photoactivation volumes and, in the case of 2PE, a dispersive optical setup for temporal focusing to control and localize the illumination pattern in the axial direction.

14. **Spr-2 suppresses sel-12 presenilin mutations by a novel mechanism**
Bernard Lakowski, Paris Pasteur

15. **Polymer scaffold seeded with stem cells – a tool for spinal cord injury repair**
Pavla Jendelova, Institute of Experimental Medicine, Prague

16. **Fast and slow self-inhibition of cortical neurons**
Alberto Bacci, EBRI Rome

17. **GDNF-dependent mechanisms reuquired for catecholaminergic neuron survival**
    María Hildalgo-Figueroa (PhD in Alberto Pascual's group), University Seville

18. **3D-organization of the presynaptic T-bar in Drosophila neuromuscular junction**.
    Oleg Shupliakov, KI Stockholm

19. **Development neuroscience in the hiStone age**
    Ola Hermanson, KI Stockholm

20. **Neuronal and synaptic physiology in mouse models of mental retardation**
    Yann Humeau, INCI Strasbourg

21. **Proteolytic shaping of synapses**
    Leszek Kaczmarek, NENCKI Warsaw

22. **Gaining Information from the Time Domain of [Ca2+] - Signals**
    Detlev Schild, University Göttingen

Posters

1. **Gene Expression profiling of cortical progenitors in ferret**
   Camino de Juan Romero, Neuroscience Institute of Alicante

2. **Evidences for the widespread existence of OSVZ in the developing cerebral cortex among gyrated mammals**
   Isabel Reillo Cuesta, Neuroscience Institute of Alicante

3. **Zic2 controls eye-specific refinement at visual targets by directly regulating the expression of the serotonin transporter**
   Cristina Carcia Frigola, Neuroscience Institute of Alicante

4. **Neuronal networks in separate layers of the prefrontal cortex are differentially modulated by nicotinic receptors**
   Rogier Poorthuis, CNCR, Neuroscience campus Amsterdam, VU Amsterdam

5. **Role of Cyclin-Dependent Kinase 5 Phosphorylation of Munc18 in synaptic transmission**
   Sabine K. Schmitz, CNCR Amsterdam

6. **Role of Synaptotagmin 1 in Large Dense Core Vesicle dynamics and release**
   Tony Cijsouw, CNCR, VU Amsterdam
7. **Molecular machinery for dense core vesicle secretion in neurons**
   Ruud Toonen, CNCR, VU Amsterdam

8. **Adolescent nicotine exposure induces lasting changes in presynaptic mGluR2 function in excitatory synapses in prefrontal cortex**
   Natalia A Goriounova[1], Danielle S Counotte[2], Sabine Spijker[2], August B Smit[2], Huibert D Mansvelder[1]
   [1] Department of Integrative Neurophysiology, Center for Neurogenomics and Cognitive Research, Vrije Universiteit, Amsterdam, The Netherlands; [2] Department of Molecular and Cellular Neurobiology, Center for Neurogenomics and Cognitive Research, Vrije Universiteit, Amsterdam, The Netherlands

   Adolescence is a time period of increased vulnerability for developing drug addiction, with the majority of adult smokers starting the habit at this age. Brain maturation is not complete at adolescence, and in particular the prefrontal cortex (PFC) continues developing during this period, as does cognitive performance. In rodents, nicotine exposure during adolescence results in decreased attention performance and augmented impulsivity during adulthood, suggesting reduced prefrontal function. Here, we asked the question whether adolescent nicotine exposure induces lasting effects on the adult prefrontal cortex neuronal circuitry and function. We used a combination of synaptic proteomics and electrophysiological techniques to study the mechanisms underlying the cognitive deficits resulting from adolescent nicotine exposure. We find that nicotine injections during adolescence (P34-43) affect the levels of only a handful of synaptic proteins in PFC of adult rats, and most prominently reduced levels of synaptic metabotropic glutamatergic receptor 2 (mGluR2) protein. This reduction in receptor levels is paralleled by diminished presynaptic mGluR2 modulation of excitatory transmission in layer V pyramidal neurons. Activation of mGluR2 receptors in these neurons leads to decreased evoked EPSCs, reduced frequency of mEPSCs and increased paired-pulse ratio, indicating that mGluR2s act presynaptically by inhibiting glutamatergic transmission. In conditions of high glutamate release such as during high frequency stimulation mGluR2 action would be most prominent. Accordingly, we find that adolescent nicotine exposure also leads to reduced short-term depression at excitatory synapses suggesting decreased filtering of excitatory inputs necessary for selective attention. Thus, nicotine exposure during adolescence results in lasting reduction in presynaptic mGluR2 signaling and short-term plasticity in adult PFC, implicating the role of these adaptations in lasting cognitive deficits at adult age.

9. **The role of the PICK1-Arf1 interaction in regulating AMPA receptor traffick**
   Dan Rocca, University of Bristol

10. **The role of SUMOylation in Kainate Receptor trafficking during Oxygen Glucose Deprivation**
    Siobhan Dennis, University of Bristol

11. **Gating of Synaptic Plasticity by Muscarinic M 1 Receptors in the Hippocampus**
    Sophie Chamberlain, University of Bristol

12. **Identification and characterization of proteins interacting with GPCR Interacting Scaffold Protein (GISP)**
    Sriharsha Kantamneni, University of Bristol

13. **Examining the role of Notch-signalling in adult hippocampal stem cell maintenance**
    Oliver Ehm, Helmholtz Center Munich

14. **XtraCOunt: a novel semi-automatic tool for investigating synaptic vesicle localisation**
    Christian Olendrowitz, ENI Göttingen

    The novel stand-alone desktop application XtraCOunt, an image analysis tool, can be used to semi-automate the data acquisition of electron micrograph based synaptic vesicle distribution studies. With the help of the user-friendly application the analysis of single images is performed within minutes, in a fraction of the time spend previously.
    Furthermore, the enormous variability for measurements and output formats turns XtraCOunt additionally into an easy-to-use tool, which is making image analysis studies across a wide range of scientific fields, finally more feasible, more comparable and less biased.

15. **Dissecting the role of the ARF guanine nucleotide exchange factor GBF-1 for Golgi transport in *C. elegans***
    Mandy Hannemann, ENI Göttingen

16. **The Anaphase-Promoting Complex in Synaptice Function, Learning and Memory**
    Tanja Kuczera, ENI Göttingen

17. **Role of Formin in extinction of fear memory**
    Cemil Kerimoglu, ENI Göttingen

18. **Fast but not slow temporal patterning improves odor coding in the olfactory bulb**
    Olivier Gschwend, University of Geneva, Department of Neurosciences

19. **Transient Neuronal populations are necessary for the formation of the Corpus Callosum**
    Mathieu Niquille, DMCB-UNIL Lausanne

20. **Investigating cortical spiking dynamics *in vivo* using simultaneous intracellular and multiunit recordings**
    Lisa Beeren, UC London

21. **Inhibition in the granule cell layer of the cerebellum**
    Daniel R Ward, Roby T Kanichay & R Angus Silver, Department of Neuroscience, Physiology and Pharmacology, University College London, UK

    The cerebellum integrates mossy fibre (MF) and climbing fibre information in the Purkinje cell layer to perform a variety of computations relating to movement, motor learning, balance and cognition. Before MF

information reaches the Purkinje cells, it passes through the granule cell (GrC) layer where it is transformed, in part by a local inhibitiory circuit.
We have electrophysiologically characterised the inhibitory input of GrCs in young adult (P25-35) Sprague Dawley rats. We show that GrCs recieve two distinct flavours of inhibition: Phasic inhibition, resulting from synaptic release of gamma-aminobutyric acid (GABA) from specialised Golgi cell (GoC) terminals onto GrC post-synaptic densities containing GABA type A receptors ($GABA_ARs$). And tonic inhibition from the constitiutive activation of high affinity $GABA_ARs$ at extrasynaptic sites by ambient GABA concentrations.
We demonstrate that tonic inhibition provides the majority of the inhibitory conductance at rest. However, due to "spillover" of phasically released GABA onto higher affinity extra-synaptic sites, sustained activation of an average GoC input at physiological rates (20 – 50 Hz) generates an additional steady conductance roughly one third the size of the tonic. Previous studies have suggested that each GrC recieves ~8 GoC inputs, as such, phasic inhibition could represent the dominant means of limiting MF information flow *in vivo*. Conversely, glutamate released by local MF activity can limit the magnitude of GoC phasic inhibitory input by activating presynaptic metobotrophic glutamate receptors.

22. Cone dystrophy with supernormal electroretinogram: Functional analysis of missense mutations in *KCNV2* that encodes the voltage-gated potassium channel subunit, Kv8.2

Katie E Smith[1,2], Susan E Wilkie[1], Martin Stocker[2] and David M Hunt[1], [1]UCL Institute of Ophthalmology, 11-43 Bath Street, London EC1V 9EL, UK and [2]UCL Research Department Neuroscience, Physiology and Pharmacology

Cone dystrophy with supernormal electroretinogram (CDSE) is an autosomal recessive condition resulting in a progressive loss of visual acuity and colour vision combined with a delayed scotopic and photopic response to light flashes and an enhanced b-wave at high flash intensities. Recently, homozygosity mapping identified *KCNV2* as the causative gene. *KCNV2* encodes a voltage-gated potassium (Kv) channel subunit, Kv8.2, which incorporates a tetramerisation (T1) domain at the intracellular N-terminal, six transmembrane domains (S1-6), and a pore region containing the potassium-selective motif GYG. However, it does not form functional homomeric channels but acts in a modulatory fashion when combined into heteromeric channels with Kv2.1 subunits. Both Kv8.2 and Kv2.1 subunits are expressed in the inner segments of photoreceptors, which together with the unusual ERG, suggest a dysfunction affecting the first synapse of photoreception.
The mutations in *KCNV2* that have been found in patients with CDSE include in-frame deletions, frame shifts, missense, nonsense, and non-stop mutations. This study has focused on missense mutations that occur in different regions of the Kv8.2 protein.
When expressed in mammalian cells, Kv8.2 subunits are retained intracellularly, but when coexpressed with Kv2.1, Kv8.2 and Kv2.1 are incorporated into functional channels at the plasma membrane. Two of the mutations identified in patients, L126Q and W188C, occur at the intracellular N-terminal within or in close proximity to the T1 domain; yeast two hybrid experiments show that the T1 domains of Kv8.2 subunits with either of these mutations lack the ability to interact with the T1 domain of Kv2.1. Two further mutations found in patients, W450G and G459D, occur in the pore region; confocal imaging and electrophysiological experiments indicate that these mutant subunits are incorporated into channels with Kv2.1 subunits but that the resulting channels are non-functional. For both sets of mutations therefore, the ability of the mutant Kv8.2 subunit to interact with Kv2.1 and/or form functional channels is abolished, consistent with the concept that it is the absence of functional Kv8.2/2.1 channels which results in CDSE.

23. Characterisation of a Ca^{2+}-dependent afterdepolarising current in cultured hippocampal pyramidal neurons

Marisol Sampredo Castaneda and Paola Pedarzani, UC London

Neuronal firing is usually associated with transient elevations of intracellular Ca^{2+} which may in turn exert multiple effects on a variety of cellular targets. Amongst these, Ca^{2+}-activated ion channels have been subject of study for decades as their gating produces post-spike changes in membrane potential capable of shaping subsequent neuronal activity. Hippocampal pyramidal neurons express a variety of these

conductances, the most well-understood of which are underlied by Ca^{2+} activated K^+ channels and play a major role in spike frequency adaptation. Here we report the occurrence of a novel Ca^{2+} dependent mixed-cationic current in cultured hippocampal neurons. This conductance, named I_{ADP}, has a time course in the order of hundreds of milliseconds and likely interacts with the Ca^{2+}-dependent, medium duration afterhyperpolarising current mediated by SK and KCNQ channels in these neurons. The main biophysical and pharmacological features of I_{ADP} are presented and discussed with a focus on the TRP channel superfamily as possible molecular correlates for I_{ADP}.
This work has been supported by the UK Medical Research Council.

24. Neuromodulatory effects of the neuropeptide pituitary adenylate cyclase activating peptide (PACAP) on hippocampal neurons.
Marita Gronning Madsen, UC London

25. FoxO transcription factors in adult neurogenesis
Muhammad Amir Khan, Tobias Schwarz, Chichung Lie, Helmholtz Center Munich

FoxO transcription factors are involved in many aspects of cellular functions, such as metabolism, transformation, cell cycle arrest, differentiation, cell death and protection from stress stimuli. There are currently four members in this group; FoxO1, FoxO3, FoxO4 and FoxO6. FoxO transcription factors can bind to the same target sequence and can show overlapping functions. Recent studies have indicated a role for FoxO transcription factors in adult stem cell regulation. For example, FoxO1-/-, FoxO3-/-, FoxO4-/- triple conditional knock out mice revealed a significant decrease in the long-term hematopoietic stem cell population.
The adult hippocampal dentate gyrus is one out of two regions of the adult mammalian brain where neural stem cells generate new functional neurons throughout adulthood. We investigated the expression of FoxO proteins in the adult neurogenic areas in the brain. Interestingly, we found the activity of FoxO proteins in Sox2 and GFAP positive cells in the subgranular zone of the hippocampal dentate gyrus. Sox2 and GFAP positive cells represent the putative stem cells in the subgranular zone, arguing that FoxO proteins can play a role in the maintenance of quiescence in these cell types. We have also detected the activity of FoxO proteins in the immature neurons in the dentate gyrus. FoxO proteins are involved in controlling the differentiation of many cell types arguing a similar role in the new born immature neurons in the dentate gyrus. Moreover, we have found that the activity of FoxO proteins is differentially regulated in proliferating and differentiating neural stem cells in vitro. At present we are generating a GLAST creERT2:: FoxO1/3/4 conditional knockout line to investigate the role of FoxO proteins in adult neural stem cell maintenance. Finally, we have also begun to use Cre-recombinase encoding retroviruses to investigate the role of FoxO in immature neurons.

26. Boundary formation and neuronal differentiation in the zebrafish hindbrain
Niklas Senghaas, Helmholtz Center Munich / IDG / Neuroimaging Group

27. Establishment of human polyglutamine disease model using zebrafish
Dr. Kazuhiko, Helmholtz Center Munich / Institute of Developmental Genetics

28. Adaptive plasticity of neuronal circuits during postnatal mouse development
Heidi Söllner, Helmholtz Center Munich / Institute of Developmental Genetics

29. Who is leading whom? Neuroplin 1 in the development of the sensory-motor circuit
Rosa-Eva Hüttl, Helmholtz Center Munich / Institute of Developmental Genetics

30. Optical Measurements of membrane voltage in neuronal subcompartments
Ann Fink, Paris Descartes

31. Properties of excitatory synaptic transmission onto cerebellar interneurons
Therese Abrahamsson, Paris Descartes

32. Arbitrary two-photon excitation patterns by spatiotemporal shaping of ultrashort pulses

E. Papagiakoumou[1], V. de Sars[1], D. Oron[2], V. Emiliani[1]
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Multiphoton excitation has recently found application in the fields of bioimaging, photoactivation and lithography. In order to fully exploit the advantage of the axial resolution due to the nonlinear excitation, most systems to date operate by scanning either the laser beam or the sample to generate the illumination pattern. However, scanning can be proved time-consuming for applications requiring uniform excitation of large areas over short time scales, such as neuronal activation by multiphoton uncaging of neurotransmitters. Here, we present a new, scanning-less method to generate arbitrary shaped, depth resolved excitation patterns by combining the recently introduced technique of temporal focusing with recent advances in holographic patterned illumination. A liquid crystal spatial light modulator (LC-SLM) is used to control lateral extension of the excitation spot. A dispersive optical element inserted at the Fourier plane of the LC-SLM is used to control the axial resolution. We present an experimental and theoretical analysis of the effect of spatial phase patterning on the depth resolution achieved in temporal focusing microscopy and we show that an axial confinement of about 5μm (i.e. comparable to line scanning two-photon excitation) is reached, independently on the lateral extension of the excitation spot. The theoretical analysis revealed that although pure amplitude modulation and flat wave-front would lead to a better axial confinement, the use of a holographic wave-front only slightly deteriorates the axial resolution even for the limiting case of a random phase. By introducing this method, we devise new schemes for optimal illumination using temporally focused excitation.

33. Subcellular distribution of presynaptic chlorid-permeable glycine receptors

Johana Trojanova, ASCR Prague

34. Development of a live reporter system to identify CREB-activated neurons

Leiron Ferarrese, EBRI Rome

35. Characterization of hippocampal synaptic plasticity phenotype in AD11 mice

Gry Houeland, EBRI Rome

36. Neuronal adaptations induced by robust combinational antidepressant treatment in CA 1 pyramidal cells of the hippocampus

Elisiani Tafi, EBRI Rome

37. An unexpected action of the antidepressant imipramine: inhibition of GABA release by a specific hippocampal interneuron subtype

Pablo Mendez Garcia, EBRI Rome

38. Presynaptic degeneration of hippocampal interneurons in the brain of knock-out mice lacking Cysteine String Protein-alpha (CSP-alpha)

Leonardo Gomez-Sanchez, University Seville
Leonardo Gómez-Sánchez1, Pablo García-Junco-Clemente1, Gloria Cantero-Nieto1, Pedro Linares-Clemente1, Rafael Luján2 and Rafael Fernández-Chacón1
1Instituto de Biomedicina de Sevilla (IBiS) Hosp.Univ. Virgen del Rocío/CSIC/Univ. de Sevilla, Dpto. De Fisiología Médica y Biofísica Univ. de Sevilla and Centro de Investigación Biomédica en Red deEnfermedades Neurodegenerativas (CIBERNED), Avda. Sánchez-Pizjuán 4, 41009 Sevilla, Spain; 2Dpto.de Ciencias Medicas, Facultad de Medicina & Centro Regional de Investigaciones Biomédicas,Universidad de Castilla-La Mancha, 02006 Albacete,Spain

The maintenance of high activity regime in many synapses probably requires molecular mechanisms that recycle unfolded or damaged proteins. Cysteine String Protein-alpha (CSP-alpha) is unique among synaptic vesicle proteins because it contains a DNAJ domain characteristic of HSP40 co-chaperones. Since CSP-alpha KO mice display early presynaptic degeneration, CSP-alpha has been proposed as a molecule involved in the protection of nerve terminals. We are interested in clarifying if the CSP-alpha dependentphenotype is related to presynaptic activity. In hippocampal cultures lacking CSP-alpha, we have found a progressive reduction in the number of GABAergic synapses but no changes in the number of glutamatergic terminals compared to control cultures. Remarkably, knock-out cultures were devoid of interneurons expressing parvalbumin(PV) and synapses expressing synaptotagmin2. Since those proteins are hallmarks of fastspiking hippocampal basket cells, our observation suggest that those neurons particularly require CSP-alpha. Then, we have followed-up our observations directly in the mouse brain. Our initial data indicate that mice lacking CSP-alpha progressively losesynaptotagmin2-expressing synapses but they still maintain a normal number of PVpositiveneurons. Probably, the absence of CSP-alpha initially leads to degeneration ofbasket cells presynaptic terminals that triggers a dying back process that is, however, faster in culture than within the physiological hippocampal niche in the brain.

38a CSP-alpha is differentially required by GABAergic and glutamatergic hippocampal synapses to maintain synaptic function
Rafael Fernandez-Chacon, University Seville

39. Understanding the adult carotid body stem, cell niche
Aida Platero-Luengo, Rocío Durán, José López-Barneo and Ricardo Pardal, University Seville

Recent observations in our lab identified the mammalian carotid body (CB) as a niche of neurogenesis with a recognizable physiological function in adult life.This organ is responsible for the detection of hypoxemia and is able to adapt to a persistent stimulus by increasing the number of neuronal, type I cells. Wehave previously shown that this neurogenesis depends on the proliferation and differentiation of a subpopulation of glia-like stem cells or type II cells. However, the specific mechanisms by which CB stem cells (CBSCs) are activated in
hypoxia are unknown. We hypothesize that blood vessels and CB neuronal cells might have an important role on instructing the stem cells to proliferate and differentiate. To elucidate this mechanism we are using different methodological approaches: 1.- In vitro cultures to test the effect of different niche factors on the proliferation and differentiation of CBSCs; 2.- PCRs to study the expression of specific receptors and signalling factors in the different CB cell types separated by flow cytometry; 3.- Electron microscopy to study the cytoarquitecture of the
CB niche, and immunochemistry approaches to reveal the expression of
specific proliferation and differentiation markers in vivo. Understanding the physiology of CBSCs is crucial not only to learn about adult neurogenic niches but also to improve the use of these cells for therapeutics and to better understand the pathophysiology of the organ.

40. Thyrotropin-Releasing Hormone regulates Intra-Arcuate Rhythmicity
Christian Broberger, KI Stockholm

41. Rhythmic oscillation in tuberoinfundibular dopamine (TIDA) neurones are switched to tonic firing by thyrotropine-releasing hormone (TRH): A novel principle for the control of prolactin secretion.
David Lyons, KI Stockholm

42. The role of dynamin-endophilin complex in servering membranes during synaptic vesicle recycling
Anna Sundborger, Nikolay Tomilin, Jenny Hinshaw and Oleg Shupliakov, KI Stockholm

Recycling of synaptic vesicles after neurotransmitter release is crucial for sustained transmission and this membrane retrieval process occurs mainly through the clathrin-mediated endocytosis pathway. The GTPase Dynamin is believed to mediate fission of newly formed vesicles from the plasma membrane at the last

stage of this process, though the exact mechanism of its function is unknown. Dynamin contains a Plecktrin-homology domain, which interacts with membrane lipids, and a proline-rich domain that enables it to bind SH3 (Scr homology 3) domains. Several SH3-domain containing proteins are enriched in the synapse and are known to bind Dynamin *in vitro*. One of them is Endophilin, which binds Dynamin via its C-terminus SH3 domain. Endophilin exists as a dimer in solution and forms a lipid interacting module with its N-terminus BAR domains. Both Endophilin and Dynamin tubulate pospholipids and accumulate on the lipid tubes when mixed together. Our present study elucidates the role of Endophilin in regulation of the Dynamin function in fission.

To address this question we first used synapses established by giant reticulospinal axons in lamprey (*Lampetra fluviatilis)* to localize both proteins at sites of synaptic vesicle recycling (periactive zones) and to study the effects of perturbation of the interaction between Dynamin and Endophilin on endocytosis *in situ*. Using immunogold imunocytochemistry we show that Endophilin is accumulated at necks of clathrin-coated pits in intact synapses along with Dynamin. We used the synaptojanin derived proline-rich peptide (pp19) and antibodies blocking the interaction between the two proteins to study the effects on endocytosis. Ultra-structural analysis of the effects of these compounds showed that endocytosis was inhibited, but not blocked. Further, Endophilin and Dynamin assembled into a visible complex at necks of coated pits in stimulated synapses in the presence of GTPγS (a non-hydrolysable analogue of GTP). This assembly was blocked by the application of pp19. Injection of the SH3 domain of Endophilin also resulted in inhibition of fission. In these synapses, Dynamin assembled into spirals, but these lacked attachment to the membrane necks.

In vitro studies with recombinant proteins showed that Endophilin and Dynamin could assemble on liposome templates and form very thin tubes decorated by spirals. This assembled spiral was structurally different to what can be observed when proteins are added alone to liposomes, (ones when proteins were applied alone) thus suggesting that they form a complex. Under

conditions that prevent efficient tubulation and decoration of liposomes by Dynamin, the presence of Endophilin caused an increase in formation of Dynamin decorated tubes. Thus, our results indicate that Endophilin promotes the recruitment of dynamin to the neck of coated pits and the assembly of the fission complex during endocytosis.

43. Lateral hypothalamic TRH neurones, a potential link between the central metabolic senso and cortical arousal
Emilia Horjales, KI Stockholm

44. Total and conditional deletion of αGDI protein suggests causal relationship between cortico-amygdala pathway and aggressive behaviour
Frédéric Gambino, CNRS Strasbourg

45. The stimulation of beta2 adrenergic reports relieves allodynia in a model of neuropathic pain in mice
Nada Choucair, INCI Strasbourg

46. Multiplication of beta-2 adrenoceptors in the antiallodynic action of antidepressant treatment in neuropathic pain
Yalcin-Christmann Ipek, INCI Strasbourg

47. Manipulation of Ttyh1 gene expression influences cell morphology in neuroblastoma and hippocampal neurons in vitro
Marzena Stefaniuk, NENCKI Warsaw.

48. PICK 1: A novel regulator of spine morphology
Yasuko Nakamura, University of Bristol

49. UNC-50 and GARP complex function redundantly in membrane trafficking
Luo Ling, ENI Göttingen

50. A new transgenic mouse model to study gliotransmission in vivo

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The development and function of neurons in the central nervous system (CNS) and their demise under pathological conditions are rarely cell-autonomous and depend often on interactions with glial cells. Molecules that are released by glial cells play a key role in these interactions: they influence the generation and differentiation of neurons, modulate synaptic transmission and influence neurodegeneration. We aimed to test the long-standing hypothesis that exocytotic release from astrocytes influences synaptic function. To accomplish this, we have generated new lines of transgenic (Tg) mice that allow for temporally controlled block of SNARE-dependent exocytosis from astrocytes. This approach is based on the Cre recombinase-dependent induction of the light chain from the Clostridial neurotoxin Botulinum serotype B (BoNT/B). This toxin cleaves synaptobrevin/VAMP2, an essential component of the SNARE complex. To visualize cells undergoing recombination, the trangenic construct also contain EGFP sequence. First, we aimed to validate our model by crossing our toxin line with Tg mice, where Cre is controlled by a ubiquitous promoter CMV. This should mimick the phenotype of the synaptobrevin/VAMP2 knockout, which is lethal on postnatal day 1. Among 46 births, 28% wt and 64% single Tg mice were observed, but no viable double Tg animals. We observed frequently dead pups, which were all double-Tg when they could be tested. To target the toxin to astrocytes, we crossed the toxin line with new Tg (Tg) lines that permit somatic mutagenesis in sub-types of astrocytes. In these mice, Cre recombinase expression is controlled by the Glast (line T45-72) or Connexin 30 promoter (T53-33, Slezak et al. 2007, Glia). To study the relevance of calcium- SNARE-/dependant exocytosis in vivo, we focussed on changes in the retina. We used a highly sensitive biochemical assay based on supraplasmatic resonance measurements (Ferracci et al. 2005, Biochem J) to detect VAMP in tissue lysates and to test whether retinae from adult double Tg lines Tg(Glast-CreERT2) x Tg(CAG-iBotB) contained toxin activity after tamoxifen (TAM) administration. We found that the level of VAMP2 normalized to synaptophysin was reduced. We then tested whether toxin induction in Müller cells affected retinal function using electroretinography. We found that ERGs of TAM-injected double Tg mice were very similar to those of TAM-injected monogenic mice. Classic histological staining revealed no drastic changes in global morphology 1 or 3 weeks after TAM-induced recombination. Double Tg animals show a slight but significant increase in food intake without changes in body weight compared to control animals when monitored for 16 weeks after TAM injection, but there was no difference in global locomotor activity between the two groups. Taken together, our results indicate that the Tg approach allows to block exocytosis in cells that can be targeted by Cre lines. The block of exocytosis in Müller glia did not affect retinal function or morphology.

51. Prediction and experimental verification of miRNA-mRNA interaction related with synaptic plasticity

Nestor Karathanasis, IMBB-FORTH Crete

52. Investigating the link between late-onset neurodegeneration and the ageing process

Manolis Vlachos and Nektarios Tavernarakis, IMBB-FORTH Crete

C. elegans is a powerful model organism for investigating the molecular mechanisms underlying important biological processes such as ageing and neurodegeneration. Ageing is associated with several neurodegenerative disorders (e.g. Alzheimer's, Parkinson's and Huntington diseases). Many mutations affecting known longevity pathways have been shown to delay age-related pathologies. However, the mechanisms that link the ageing process with late-onset neurodegeneration are not well-understood. To address this question, we are analyzing the effects of lifespan-extending mutations on necrotic neuronal degeneration in *C. elegans*. We have examined the potential role of major, evolutionary conserved, longevity pathways (insulin/IGF-1 signaling, caloric restriction, protein synthesis) on neurodegeneration induced by hyperactive ion channels. In a parallel approach we are studying the role of lysosomal

biogenesis and acidification, which have been implicated in necrotic cell death, on *C. elegans* ageing. Lysosomes are low-pH membrane bound organelles which have a degradative role within cells and function in diverse cellular processes. To examine their role in *C. elegans* ageing, we are targeting genes known to regulate lysosomal biogenesis and acidification and assessing their effects on organism lifespan. Genes that encode either for cytoplasmic or for transmembrane subunits of the *C. elegans* vacuolar H^+-ATPase complex which pumps protons into lysosomes (maintaining their low luminal pH), are among the genes included in our study. Furthermore, we are also examining the possible effect of mutations that interfere with the biogenesis and distribution of gut granules (a class of intestine-specific acidified lysosome-related organelles), and have been previously shown to affect necrosis, on *C. elegans* ageing.

53. Small heat shock proteins protect against necrotic cell death

Nikos Kourtis, IMBB-FORTH Crete

Necrotic cell death contributes to severe pathological conditions in humans such as trauma, stroke and neurodegenerative diseases. The molecular mechanisms underlying necrosis are not fully understood. The heat shock response is a highly conserved gene expression program, which is engaged under conditions of stress and orchestrates the coordinated expression of specific genes that protect cells against various stressors. We are investigating the role of the heat shock response in necrotic cell death. We find that activation of the heat shock response pathway by means of a brief heat shock treatment strongly suppresses necrotic cell death in *C. elegans*. This protective effect is not due to delay of necrosis initiation or removal of the necrosis initiating insult. Elimination of heat shock factor 1 (HSF-1), the master transcription regulator which orchestrates the heat shock response in *C. elegans*, abolishes the protective effect of heat shock. By contrast,
overexpression of HSF-1 suppresses necrosis. While screening for potential mediators of the protective effect of heat shock, we found that the genes encoding for the small heat shock proteins HSP-16.1 and HSP-16.48 are specifically required for the protective effect of heat shock on necrosis. Moreover, overexpression of HSP-16.1 provides protection against necrotic cell death and circumvents the requirement for heat shock response activation. Further characterization of the protective function of the heat shock response activation and HSP-16.1 in necrosis may facilitate the development of intervention strategies aiming to counter necrotic cell death.

54. The impact of somatic misexpression of germline feature on neuronal ageing in *Caenohabditis elegans*

M. Rieckher and N. Tavernarakis, IMBB-FORTH Crete

Aging is an omnipresent phenomenon affecting almost all organisms and characterized by degenerative changes in cells and tissues and organs, such as the nervous system, that result in a decrease in life-quality, higher susceptibility to diseases and therefore an increase in the probability of death. Our specific research interests in the biology of ageing focus on the genetic mechanisms underlying germline immortality in *C. elegans*.
The germ line proliferates indefinitely and is therefore considered an immortal cell lineage. Mutations of the *Caenorhabditis elegans* synthetic *Multivulva B* (*synMuvB*) genes lead to the appearance of germline-specific P granules and proteins in somatic cells, suggesting a reversion of the soma to patterns of germline-specific gene expression.
I am investigating ageing in *C. elegans synMuvB* mutants. The missexpression of germline features in somatic cells of these mutants is likely to promote longevity in *C. elegans*. Thus, by investigating the molecular mechanisms mediating these effects we may gain insight into fundamental distinction between germline immortality and soma.

55. Clathrin-mediated endocytosis and intracellular trafficking are required for necrotic cell death in *C. elegans*

Kostoula Troulinaki and Nektarios Tavernarakis, IMBB-FORTH Crete

Defects in endocytosis and trafficking of endocytic vesicles and organelles are apparent and have been implicated in many human neurodegenerative conditions such as Alzheimer's disease, Huntington's disease

and ALS. One of the early events in the course of neuronal necrosis in *C. elegans* is the formation of small, tightly wrapped membrane whorls that at a later stage are internalized and coalesce into large, electro-dense membranous structures, implicating mechanisms of endocytosis and trafficking in necrotic cell death (Hall et al., J. Neurosci. 17: 1033). To gain insight into the molecular mechanisms underlying these cellular events, we examined the requirement for specific endocytosis and trafficking genes in necrosis. In addition, we monitored endocytosis during cell death. We find that neurodegeneration induced either by hyperactivated ion channels or by chemical treatment is suppressed in genetic backgrounds deficient for proteins that are required for clathrin mediated endocytosis and transport of vesicles along microtubules. In addition, we find that specific endocytotic proteins and two kinesins function together with calpain cytoplasmic proteases, and lysosomal cathepsin proteases to mediate necrosis. Furthermore, endocytotic components synergize with autophagy, a process which is induced and contributes to cellular destruction. Our observations indicate that clathrin-mediated endocytosis and intracellular trafficking are important for necrotic cell death in the nematode.

56. The contactin homolog *rig-6* is involved in axon guidance and branching in *C. elegans*

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The Immunoglobulin Superfamily (IgSF) is a conserved family of proteins, playing a leading role in many developmental processes, including nervous system patterning. The contactin subgroup of the IgSF consists of glycosylphosphatidylinositol (GPI) anchored glycoproteins, known to be essential for axon growth, guidance and fasciculation, neuronal migration and myelination in vertebrates. Aiming to further understand the role of contactins, we are characterizing RIG-6 (C33F10.5), the only member of the contactin subfamily in *C. elegans.* We have determined *rig-6* spatiotemporal expression pattern; it is expressed in head neurons, ventral cord motorneurons and commissures, HSN and CAN neurons, in muscle cells, spermatheca and hypodermis. *rig-6* expression begins in embryonic stages, and it is maintained throughout adulthood.

To further investigate the role of RIG-6, we have used *rig-6* RNAi knockdown to study effects on neuronal development and axonal migration. Our data show that downregulation of *rig-6* expression leads to the formation of ectopic branches in ALM axons. Moreover, we have observed abnormal crossing of axons in the ventral nerve cord (VNC)

To over-express *rig-6,* we have generated a plasmid that encompasses the promoter, the complete coding sequence and the UTRs of the gene. Transgenic animals show cross defects in the VNC, revealing that the level of expression of *rig-6* is critical for normal axon guidance in the VNC. In addition, commissures are misguided and tend to form branches. Several behavioral abnormalities have been observed in the gain of function mutants of *rig-6*, namely in locomotion, defecation and fertility.

Mutations in UNC-53, a cytoskeleton binding protein involved in anteroposterior cell migration and axon guidance, cause the formation of ectopic branches in ALM axons, abnormal commissure branches as well as cross defects in the VNC. To test whether *unc-53* and *rig-6* function in the same pathway, we have used *rig-6* RNAi in *unc-53(n152)* mutant animals. *rig-6* downregulation enhances the formation of commissure branches in *unc-53(n152)* mutants. Moreover, *rig-6* overexpression in *unc-53(n152)* animals leads to increased frequency of commissure branches and cross defects compared to *unc-53(n152)* or *rig-6* gain of function mutants alone. Our data suggest that *rig-6* affects axon guidance and branching in *C. elegans*. UNC-53 could act downstream of RIG-6, converting an extra-cellular signal to an intracellular response.

57. Investigating the roles of protein SUMOylation in cerebral ischaemia

Helena Cimarosti, University of Bristol

58. A screen for synaptic SUMO-1 Substrates

Kevin Wilkinson, University of Bristol

59. M-current Modulation of Gamma oscillations and action Potential Phasing in the Hippocampus
Hui Min Tan, KI Stockholm

60. Orexigenic nociceptin orphanin FQ, in hypothalamic neurons associated with the control of energy balance
Nasren Maolood, KI Stockholm

61. Epiletogenesis in cyclin D2 deficient mice that are devoid of adult neurogenesis
Gabriela Plucinska, NENCKI Warsaw

62. Molecular determinants underlying the functional specification of motor neurons
Daniel Müller, David Herholz, Liang Wang and Till Marquardt, ENI Göttingen

63. Profiling the inherent vulnerability
Daniel Müller, David Herholz and Till Marquardt, ENI Göttingen

64. Role of the endocannabinoid system in associative components of extinction
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The endocannabinoid system (ECS) is known to regulate mnemonic processes and is particularly implicated in aversive memory. In fear conditioning, disruption of CB1 receptors signalling specifically impairs extinction of aversive memory, preventing the decrease of an acquired freezing response when the situation does not predict threaten anymore. Extinction is a complex phenomenon resulting from a combination of new "safety" associative learning with non-associative habituation to repeatedly presented stimuli. Recent data using acquired freezing responses to a tone suggest that the ECS is mainly involved in habituation processes. However, the dissection of associative and non-associative components of extinction is difficult in tests where this behaviour is solely indicated by a decrease of a natural reflex such as freezing. Indeed, "pure" associative or non-associative processes can result in the same decrease of freezing upon non-reinforced presentations of the tone.
To address this issue, we use active avoidance paradigm, in which mice learn to avoid a tone-signalled shock by moving into another compartment of a shuttle box. During extinction, mice learn to inhibit their escape behaviour to stay in the initial compartment during tone presentation. Extinction is then tested in a relearning session and is expressed as decreased "perseverance" of the originally acquired avoidance response. In this protocol, extinction is not merely indicated by a quantitative decrease of a fear response, but it is expressed by a qualitative change of behaviour ("go" vs. "don't go"), thereby excluding non-associative habituation. Preliminary results indicate that CB1 receptor antagonism (SR141716A, 3mg/kg, ip) in C57BL6/N mice prior to each extinction sessions results in pronounced "perseverance" of the originally acquired avoidance behaviour as compared to vehicle-treated controls. Thus, the ECS is likely involved in associative components of inhibitory learning.

65. IL1-receptor accessory protein-like 1 (IL1RAPL1) controls inhibitory networks during cerebellar development
Marie Kneib, Frédéric Gambino, Alice Pavlowsky, Henriette Skala, Stéphane Heitz, Malik Khelfaoui, Jamel Chelly, Bernard Poulain, Pierre Billuart and Yann Humeau

Abnormalities in the formation and function of cerebellar circuitry potentially contribute to cognitive deficits in humans. In the adult, the activity of the sole output neurons of the cerebellar cortex – the Purkinje cells (PCs) – is shaped by the balance of activity between local excitatory and inhibitory circuits. However, how this balance is established during development remains poorly understood. Here, we investigate the role of IL1RAPL1, a protein linked to mental retardation and autism in the development of mouse cerebellum. Using *Il1rapl1*-deficient mice, we found that absence of IL1RAPL1 causes a transient disinhibition of deep cerebellar nuclei neurons between post-natal days 10 and 14. Upstream, in the cerebellar cortex, we found developmental perturbations in the activity level of molecular layer interneurons, resulting in the premature appearance of giant GABAA-mediated inhibitory post-synaptic

currents capable of silencing PCs. We conclude that IL1RAPL1 exerts a key function during cerebellar development in establishing local excitation/inhibition balance.

66. **Parkinson's disease related genes Irk-1 and pink-1 act antagonistically in C. elegans**
Nora Wender, ENI Göttingen

67. **Membrane properties and striatal microcircuitry of striatal projection neuron Subtypes: Relation to projection target and dopamine D1 receptor expression**
Henrike Planert, KI Stockholm

68. **Cellular, morphological and synaptic properties of neurons in the lamprey striatum**
Jesper Ericsson, KI Stockholm

69. **UNC-108/RAB-2 is involved in dense core vesicle maturation in C. elegans**
Marija Sumakovic, Jan Hegermann, Stefan Eimer, ENI Göttingen

70. **The transcription factor Zic2 determines axonal laterality at the ventral spinal cord midline**
Augusto Escalante and Eloisa Herrera, UMH Alicante

71. **Differential contribution of synaptic plasticity-related transcription factors to the activity-driven neuronal transcriptome**
Eva Benito, Luis M. Valor and Angel Barco, UMH Alicante

72. **Interpreting thalamic spikes and cortical information flow in the whisker system**
Miguel Maravall, Andrea Alenda, Marta Diaz-Quesada, Manuel Molano, Stefano Panzeri

73. **Slow self-inhibition by endogenous cannabinoids is heterogeneously expressed in the neocortex**
Simone Pacioni, Silvia Marinelli, Astrid Cannich, Giovanni Marsicano, Alberto Bacci, EBRI Rome

74. **Voltage- and temperature-dependent gating of heterologously expressed channel rhodopsin2**
Thomas Chater, University of Bristol

75. **Phosporylation of PSD-95 as a Mechanism to Regulate Signalling Scaffolds**
Derya Akad, ENI Göttingen

76. **Electroporation-based method for in vitro transfection of primary neuronal cultures**
Anna Suska, ENI Göttingen