Institut de Neurociències
UNIVERSITAT DE BARCELONA

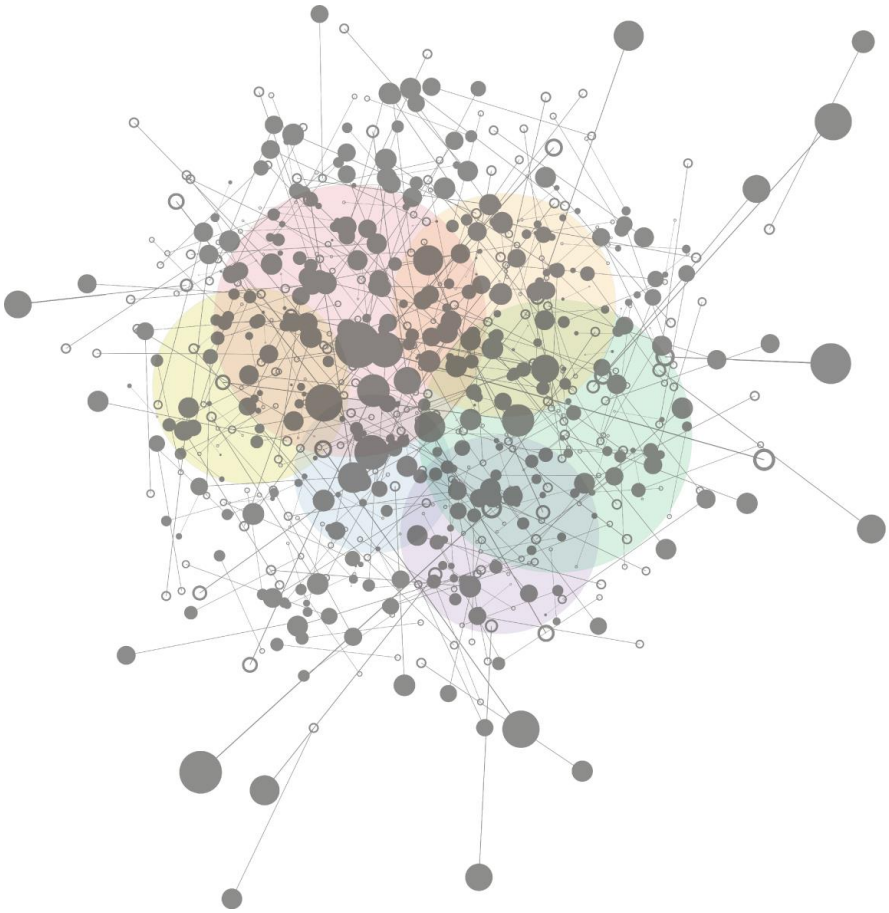
II PhD Workshop, Institute of Neurosciences

University of Barcelona

30 November - 1 December, 2017

Biology Faculty

University of Barcelona, Barcelona Knowledge Campus



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Parallel Workshops	
17:00-18:30	**Advanced Optical Microscopy applied to Neurosciences** [Room 26, Edifici Aulari] Dr. Maria Calvo and Dr. Manel Bosch, Scientific and Technological Centers; Advanced optical microscopy Unit, University of Barcelona
17:00-18:30	**Neuroimaging: Methods and Applications** [Room 14, Edifici Aulari] Dr. Carles Soriano, Bellvitge Biomedical Research Institute-IDIBELL, Psychiatry Service, Bellvitge University Hospital
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13:30-15:00	**Lunch** [Hall, Edifici Teatre]
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Plenary speaker

Molecular mechanisms of cortical interneuron diversity and plasticity

Oscar Marín
Head of the Department of Developmental Neurobiology, King's College London.

Oscar Marín was born in Madrid in 1971. He graduated in Biological Sciences from the Universidad Complutense (Madrid, Spain) in 1993, where he also obtained his Doctoral Degree in 1997 (Extraordinary award and European mention). He subsequently joined the University of California in San Francisco as a postdoctoral fellow in the laboratory of John L. R. Rubenstein. In 2003, he took a group leader position at the Instituto de Neurociencias in Alicante (Spain), a joint centre of the Spanish Research Council (CSIC) and the Miguel Hernández University (UMH). From July 2014, he is Head of the Department of Developmental Neurobiology at King's College London.

He has received numerous Awards and recognitions, such as EMBO (European Molecular Biology Organization) Young Investigator (2003), NARSAD Young Investigator (2000 and 2004), and European Young Investigator (EURYI) Awards (2004), Banco Sabadell Award for Biomedical Research (2008), Rey Jaime I Award on Basic Research (2011), the FENS/EJN Young Investigator Award (2012), the Wolfson Research Award from the Royal Society (2014), and the Prix Roger de Spoelberch (2014). He is an ERC Advanced Awardee and a Wellcome Trust Investigator.

Oscar Marín serves in several editorial boards, and he is currently a member of the Board of Reviewing Editors at Science. In 2005, Oscar Marín was selected as one of the 22 founding members of the Scientific Council of the European Research Council, where he served until 2010.

Abstract: Neural identity is thought to be established at or near the time at which neurons exit the cell cycle to become postmitotic cells, and is largely determined by intrinsic factors such as proneural genes and homeodomain proteins. Once the identity of a neuron has been established, it is generally accepted that its fate is maintained throughout life, and will not change even after heterotopic transplantation. This contributes to maintaining a stable 'ground state' that defines their role in functional circuits. Consequently, neural responses to environmental changes are thought to emerge through the function of neural circuits in which the identity of neurons remains stable. Based on these principles, the search for the mechanisms controlling the diversity of GABAergic interneurons in the cerebral cortex has primarily focused on the analysis of transcriptional programs in their progenitor cells. In this seminar, I will describe how transcriptional programmes, both during embryonic development and in the postnatal brain, regulate the identity of specific classes of cortical interneurons, thereby contributing to the generation of neuronal diversity in the cerebral cortex.

Workshops

How and why to Open Science: Grad Student Style

Asura Enkhbayar

Data Scientist, ScholCommLab, Simon Fraser University in Vancouver.

Asura Enkhbayar works as a data scientist in the #ScholCommLab at the Simon Fraser University in Vancouver, and investigates the theoretical and philosophical bedrock of Scholarly Communication as part of his PhD. Asura is an outspoken advocate and supporter of Open Science. His efforts included the participation in the Open Access Network Austria (OANA) working group “Open Access and Scholarly Communication” which published the The Vienna Principles: A Vision for Scholarly Communication in the 21st Century. Furthermore, he has been a developer and member of Open Knowledge Maps since its foundation. Within Open* he is particularly interested in the role and importance of (very) early career researchers and students. In 2016 Asura was invited to attend the OpenCon in Washington D.C. and is part of the organizing committee for OpenCon 2017 in Berlin.

Abstract: A short introduction to Open Science and its relevance for graduate students. Let us talk about the things that Open Science provides, the things that science needs, and most importantly the question "How does it help me?" The workshop is split in two parts: (1) A brief introduction to selected topics in Open Science for early career researchers or students (~40min). (2) Scientific Scavenger Hunt: A hands-on exercise for neuroscientists to explore an unknown field of research using Open Knowledge Maps (~30min). The whole workshop is meant to be a conversational and interactive session for neuroscience students to explore the concept of Open Science and its potential benefits. Please bring a laptop for the second part of the workshop.

Advanced Optical Microscopy applied to Neurosciences

Maria Calvo

Advanced Optical Microscopy facility manager, Universitat de Barcelona.

Maria Calvo (MC) holds a doctorate in biology (2001) under the direction of Dr. Carlos Enrich. She made a stay in the lab of Dr. Alexander Sorkin at the University of Colorado (2002). Later she incorporated as head of the Advanced Optical Microscopy Unit (AOMU) of the Scientific and Technological Centers of the University of Barcelona. As head of this unit, she has a proven track record (see publications) in the development and application of advanced optical microscopy and imaging analysis technologies, such as: live cell imaging, photobleaching and photolabelling techniques for the study of molecular dynamics, 2 photon excitation microscopy, in vivo (intravital) microscopy, Second Harmonic Generation, Fluorescence Resonance Energy Transfer for the study of molecular proximity and the lipid order analysis technique with Laurdan probe. AOMU is very active in organizing and participating in training and outreach activities. This activity consists on the participation in masters of the University of Barcelona, organization of courses of analysis of images, symposia in collaboration with companies, stays for degree students, participation in courses of other institutions visits for primary and secondary schools.

At the national level, MC has co-founded and coordinates the Spanish Advanced Optical Microscopy Network, obtaining a project to finance the network and coordinating the organization of the first Conference of the Spanish Network for Advanced Optical Microscopy. AOMU is currently part of the Eurobioimaging B-LIvIN Node www.eurobioimaging.eu (UB, IRB-Barcelona, CRG) and part of COST Action CA15124- New Network of European BioImage Analysts to advance life science imaging.

Manel Bosch

Advanced Optical Microscopy facility manager, Universitat de Barcelona.

Manel Bosch holds a doctorate in Developmental Biology (2007) in the University of Barcelona. He is the Advanced Optical Microscopy facility's manager with 10 years of experience. During this period, he has been involved in several scientific projects from different biomedical fields all them related to optical microscopy. He is interested in image processing and analysis. He has been training scientists in optical microscopy and image analysis and developing several protocols and SOPs.

Abstract: In the present workshop we will overview the main applications of Advanced Optical Microscopy in Neurosciences ranging from classical and robust techniques to the new ones. We will also explore how all these imaging techniques converge in the crucial step of Image Analysis to extract relevant and descriptive data from our images.

Neuroimaging: Methods and Applications

Carles Soriano

I graduated in Psychology from the Universitat Autònoma de Barcelona, and after that I obtained a master's degree in Clinical Neuropsychology and a PhD in Neurosciences. I am currently leading the Laboratory of Neuroimaging and Mental Health at the Department of Psychiatry of Bellvitge Biomedical Research Institute (IDIBELL), in Barcelona, where I directly supervise a group of eleven people (students and PhD and postdoctoral fellows). I have a wide background in structural and functional neuroimaging of psychiatric disorders. My current research interests (funded, as PI, by national and international institutions) include the assessment, through functional neuroimaging, of fronto-subcortical networks across different psychiatric disorders and the development of therapeutic tools for mental health disorders based on functional neuroimaging.

Abstract: In this workshop I will present the most important neuroimaging methods for the assessment of brain structure and function, including different sequences based on the use of magnetic resonance imaging and metabolic assessments with positron emission topography. I will also introduce the principles of imaging data processing and analysis, as well as the different analyses approaches that may be applied to the different imaging modalities and that provide multimodal information about the brain in healthy and pathological conditions.

Oral presentations

Leukoencephalopathy-causing CLCN2 mutations are associated with impaired Cl- channel function and trafficking

Héctor Gaitán-Peñas[1,2],*, Pirjo M Apaja[3,4,5],*, Tanit Arnedo[1,2], Aida Castellanos[6], Xabier Elorza-Vidal[1,2], David Soto[6], Xavier Gasull[6], Gergely L Lukacs[3,4], Raúl Estévez[1,2].

[1]Unitat de Fisiología, Departament de Ciències Fisiològiques, IDIBELL-Institute of Neurosciences, Universitat de Barcelona, L'Hospitalet de Llobregat, Spain. [2]Centro de Investigación en Red de Enfermedades Raras (CIBERER), ISCIII. [3]Department of Physiology and 4-Research Group Focused on Protein Structure, McGill University, Montréal, Quebec H3E 1Y6, Canada. [5]South Australian Health and Medical Research Institute, Nutrition and Metabolism Theme and EMBL Australia, 5000 Adelaide, Australia. [6]Neurophysiology Laboratory, Physiology Unit, Department of Biomedicine, Medical School, Institute of Neurosciences, IDIBAPS, University of Barcelona, Barcelona, Spain. *These authors contributed equally to this study.

Mutations in CLCN2 have been recently identified in patients suffering from a type of leukoencephalopathy involving intramyelinic oedema. Here, we characterised most of these mutations, which reduce the function of the chloride channel ClC-2 and impair its plasma membrane (PM) expression. Detailed biochemical and electrophysiological analyses of the Ala500Val mutation revealed that defective gating and increased cellular and PM turnover contributed to defective A500V-ClC-2 functional expression. Co-expression of the adhesion molecule GlialCAM, which forms a tertiary complex with ClC-2 and megalencephalic leukoencephalopathy with subcortical cyst 1 (MLC1), rescued the functional expression of the mutant by modifying its gating properties. GlialCAM also restored the PM levels of the channel by impeding its turnover at the PM. This rescue required ClC-2 localisation to cell-cell junctions, since a GlialCAM mutant with compromised junctional localisation failed to rescue the impaired stability of mutant ClC-2 at the PM. Wild-type, but not mutant ClC-2 was also stabilised by MLC1 overexpression. We suggest that leukodystrophy-causing CLCN2 mutations reduce the functional expression of ClC-2, which is partly counteracted by GlialCAM/MLC1-mediated increase in the gating and stability of the channel.

Pharmacological inhibition of JNK1 by Licochalcone A leads to neuroprotection

Oriol Busquets[1,2,3,4], Miren Ettcheto[1,2,3,4], Ester Verdaguer[3,4,5], Rubén Dario[2,5,6], Carme Auladell[3,4,5], Carlos Beas-Zarate[6], Jaume Folch[1,3] and Antoni Camins [2,3,4].

[1] Departament de Bioquímica i Biotecnologia, Facultat de Medicina i Ciències de la Salut, Universitat Rovira i Virgili, Reus, Tarragona, Spain. [2] Departament de Farmacologia, Toxicologia i Química Terapèutica, Facultat de Farmàcia i Ciències de l'Alimentació, Universitat de Barcelona, Barcelona, Spain. [3] Biomedical Research Networking Centre in Neurodegenerative Diseases (CIBERNED), Madrid, Spain. [4] Institut de Neurociències, Universitat de Barcelona, Barcelona, Spain. [5] Departament de Biologia Cel·lular, Fisiologia i Immunologia, Facultat de Biologia, Universitat de Barcelona, Barcelona, Spain. [6] Departamento de Biología Celular y Molecular, C.U.C.B.A., Universidad de Guadalajara y División de Neurociencias, Sierra Mojada 800, Col. Independencia, Guadalajara, Jalisco 44340, Mexico.

The mitogen-activated protein kinase family (MAPK) is a group of enzymes involved in cellular responses. One of the members of this family is the c-Jun-N-terminal kinases (JNK). The activation of the JNKs has been largely associated with the pathogenesis that occurs in epilepsy and neurodegeneration. Kainic acid (KA) administration in rodents is an experimental approach that induces status epilepticus (SE) and replicates many of the phenomenological features of human temporal lobe epilepsy (TLE). Recent studies in our group have evidenced that the absence of the JNK1 gene has neuroprotective effects against the brain damage induced by KA, as it occurs with the absence of JNK3. The aim of the present study was to analyse whether the pharmacological inhibition of JNK1 by Licochalcone A (Lic-A) had similar effects and if it may be considered as a new molecule for the treatment of SE. In order to achieve this objective, we pre-treated animals with Lic-A previous to an intraperitoneal injection of KA.

Our results showed that JNK1 inhibition by Lic-A, before KA administration, caused a reduction in the SE. Furthermore, it reduced phosphorylation levels of the JNK, as well as its activity. In addition, Lic-A prevented hippocampal neuronal degeneration, increased pro-survival and anti-apoptotic mechanisms, reduced pro-apoptotic biomarkers, decreased cellular stress and neuroinflammatory processes. Thus, our results demonstrate that Lic-A is an effective molecule on the inhibition of JNK1 and, that therefore it can be used to prevent brain damage induced by excitotoxicity.

Effects of deviant probability on middle-latency correlates of deviance detection

Fran López-Caballero, Katarzyna Zarnowiec, Carles Escera.

[1]Institute of Neurosciences, University of Barcelona, Catalonia-Spain. [2]Cognitive Neuroscience Research Group, Department of Clinical Psychology and Psychobiology, University of Barcelona, Catalonia-Spain.

Deviance detection is a key functional property of the auditory system that allows pre-attentive discrimination of novel stimuli in a context of constant stimulation, and proves that regularities in the auditory scene are encoded in the auditory pathway. Using simple-feature stimulus deviations, regularity encoding and deviance detection have been reported in brain responses at multiple latencies of the human Auditory Evoked Potential (AEP), such as the Mismatch Negativity (MMN; peaking at 100-250 ms from stimulus onset) and Middle-Latency Responses (MLR; peaking at 12-50 ms), generated at progressively lower anatomical structures in the auditory pathway. More complex levels of regularity violations, however, are only processed at higher stages of the auditory system, suggesting a hierarchical organization in auditory processing. The aim of the current study is to further characterize the auditory hierarchy of deviance responses, by assessing the sensitivity of MLR to deviant probability manipulations. MMNs and MLRs were recorded in 24 healthy subjects, using an oddball location paradigm with three different deviant probabilities (5%, 10% and 20%), and a reversed-standard (91.5%). We analyzed differences in the MLRs elicited to each of the deviant stimuli and the reversed-standard, as well as within deviant stimuli. Our results confirmed deviance detection at the level of both MLRs and MMN. Significant differences for deviant probabilities were found only for the MMN. These results suggest a functional dissociation between regularity encoding and the encoding of the probability with which this regularity is disrupted, which is only processed at higher stages of the auditory hierarchy.

Association between white matter tracts and symptomatic profiles in Huntington's disease

Clara Garcia-Gorro[1,2], Adam Hampshire[3], Richard Daws[3], Eyal Soreq[3], Joanna Sierpowska[1,2], Joan Orpella[1,2], Estela Camara[1,2,4], Ruth de Diego Balaguer[1,2,4,5].

[1] Department of Cognition, Development and Educational Pscyhology, University of Barcelona, Spain. [2] Cognition and Brain Plasticity Unit, IDIBELL (Institut d'Investigació Biomèdica de Bellvitge), L'Hospitalet de Llobregat (Barcelona), Spain. [3] Division of Brain Sciences, Department of Medicine and Centre for Neurotechonology, Imperial College London. [4] The Institute of Neurosciences of the University of Barcelona, Barcelona, Spain.. [5] ICREA (Catalan Institute for Research and Advanced Studies), Barcelona, Spain

Huntington's disease (HD) is a neurodegenerative disease that involves a triad of motor, cognitive and psychiatric disturbances. There is great variability regarding the prominence and evolution of each type of clinical sign. One possible source of phenotypic heterogeneity could be the more prominent degeneration of specific brain circuits.

The aim of the present study was to study the relationship between the different clinical symptoms of HD and the microstructure integrity of different white matter tracts. We used a multivariate approach, Canonical Correlation Analysis (CCA) that allows to study the relationship between two sets of variables.

The behavioural measures that characterized each clinical domain consisted of a compilation of questionnaires and neurological, neuropsychological and psychiatric assessments. Nineteen white matter tracts were virtually segmented using diffusion-based deterministic tractography. Diffusion indices were extracted and radial diffusivity (RD) was chosen a measure of microstructural integrity. Age was included as a nuisance variable in order to exclude their effects.

Three CCAs were carried out, one for each symptomatic domain. Regarding the cognitive domain, one significant covariance mode was found ($p = .030$) and 14 of the 19 white matter tracts showed a significant correlation with the cognitive domain. Regarding the motor domain, the significance of the mode was $p = .073$ and 11 tracts correlated with the motor domain. In contrast, in the psychiatric domain no mode of covariance approached significance.

These results show a great overlap in the white matter tracts related with cognitive and motor domains. This lack of specificity suggests that cognitive and motor disabilities could have common neurobiological basis in HD. Psychiatric symptoms, on the other hand, did not show any relation with the microstructure of the white matter tracts studied. Other tracts could be related with this type of symptoms. Alternatively, it could be possible that psychiatric symptoms arise from functional connectivity alterations.

Phenotype and genotype of negative symptoms in schizophrenia

G. Mezquida[1], B. Cabrera[1,2], M. Bioque[1,2], A. Lobo[2,3], A. González-Pinto[2,4,5], A. Espliego[6], I. Corripio[2,7], M. Bernardo[1,2,8,9]; PEPs Group.

[1]Barcelona Clinic Schizophrenia Unit (BCSU) Instituto de Neurociencias. Hospital Clinic de Barcelona. [2]CIBERSAM. [3]Instituto de Investigación Sanitaria de Aragón (IIS Aragón). University of Zaragoza. [4]Araba University Hospital, Bioaraba Research Institute, Spain. [5]University of the Basque Country (UPV-EHU), Spain. [6]Child and Adolescent Psychiatry Department, Hospital General Universitario Gregorio Marañón, School of Medicine, Universidad Complutense, Madrid. [7]Department of Psychiatry, Institut d'Investigació Biomèdica-Sant Pau (IIB-SANT PAU), Hospital de la Santa Creu i Sant Pau, Barcelona; Universitat Autònoma de Barcelona (UAB). [8]IDIBAPS. [9]University of Barcelona, Spain.

The present work aims to update the negative symptoms of schizophrenia with regard to their conceptualization, characterization and prediction through genetic and clinical markers.

The objectives of this work arose from the need, in the clinical as well as the research field, for a better and greater approach to this symptomatology. Schizophrenia is a disorder in which there hasbeen considerable progress in improving pharmacological treatments to alleviate the positive symptomatology. However, the role of pharmacological and psychological treatments for the negative symptoms has been much more limited, with little impact on these symptoms.

In recent years, genetic, inflammatory, immunological and neurotrophic markers have been studied in order to predict this symptomatology and its evolution. Focusing on the former, it has been shown that some genetic markers and their interaction with the environment play a key role in the etiology and /or pathophysiology of schizophrenia. However, in the case of some markers, it has also been suggested that they may not play a key role in the etiology per se of the disease, but rather act as potential modulators of clinical symptomatology, such as the Val66Met polymorphism BDNF. It is this gene that the first study presented here is focused on.

Another factor of interest is the study of the evolutionary pattern of the negative symptomatology throughout the different phases of the disease, as well as the different factors that may play a role in predicting a greater severity in these symptoms in the long term. The second study presented focuses on this area.

In summary, negative symptoms should be considered a clear and primary target for early intervention in patients diagnosed with schizophrenia to ensure maximum recovery, including symptomatic, functional and psychosocial recovery.

Night Eating Syndrome: Psychopathological profile in a community sample of both sexes

Carmen Varela, José Ruiz, Adela Fusté, Ana Andrés, Carmina Saldaña.

University of Barcelona, Spain.

Night Eating Syndrome (NES) is an eating disorder especially prevalent in people with overweight and obesity. NES has been associated with psychological problems like depression, anxiety or stress. For these reasons, people with NES could appeal to intake as a coping strategy.

The purpose of this research is to study the presence of NES in a community sample. Analyze the association between NES and the psychological variables anxiety, depression and eating behavior according to BMI.

A community sample of 1764 subjects was obtained via the internet: 66.4% were normalweight (NW), 22.1% were overweight (OW) and the 11.6% obesity (OB). The following questionnaires were used: Night Eating Questionnaire (NEQ) to evaluate SIN; Depression Anxiety Stress Scale in its 21-item short version (DASS-21) for assessing mood, anxiety, and stress; Dutch Eating Behavior Questionnaire (DEBQ) for the evaluation of different eating behaviors and an ad hoc sociodemographic questionnaire.

People with OB and OW get significantly higher scores in the NEQ than the NW group. In addition, people in the OB group obtain significantly higher scores than the NW group on anxiety and emotional intake, whereas people with NW have significantly higher scores on restrictive intake. No significant differences were observed for the other scales.

From the results obtained, we can observe the relationship between the presence of NES and its association with high levels of anxiety and the use of food as a method of coping with it. The concordance of these results with previous investigations shows the importance of taking into account the psychopathological profile of the patient with NES at the time of treatment.

Posters presentations: Physiopathology of Nervous System Diseases

1) Loss of synapses and calyceal juntions are early events during chronic ototoxucity in the mouse.

E. Greguske [1,2,] ,M. Carreres-Pons [1], B. Cutillas [1], J. Llorens [1,2,3].

[1.] Facultat de Medicina i Ciències de la Salut, Universitat de Barcelona, Barcelona. [2.] Institut de Neurociencies, Universitat de Barcelona, Barcelona. [3.] Institut d'Investigació Biomèdica de Bellvitge – IDIBELL, L'Hospitalet de Llobregat, Catalunya

Animal studies of the vestibular epithelium generally examine ototoxic effects after acute or short-term exposure, although chronic exposure causes most cases of human vestibular toxicity. The cellular and molecular events involved in the toxicity may differ considerably among the different exposure conditions. Male 129S1/SvImJ mice were exposed to 30 mM 3,3'-iminodipropionitrile (IDPN) in drinking water for 5 or 8 weeks. Vestibular function was assessed weekly using a specific behavioral test battery. Control and treated animals were killed at the end of the exposure period, or after a washout period of 10-13 weeks in order to study: ultrastructural features by transmission and scanning electron microscopies (TEM and SEM), immunofluorescence analysis by confocal microscopy, and qRT-PCR.

Vestibular dysfunction appeared progressively during exposure, and significant recovery occurred during the washout. SEM analysis revealed that intact hair bundle density and shape can associate with significant vestibular dysfunction, but that significant loss of hair cells by extrusion are associated with deeper, persistent dysfunction. The toxicity caused dismantlement of the calyceal junctions between type I hair cells (loss of caspr1 and tenascin-C; misplacement of KCNQ4) and loss of synapses (decreased numbers of ribeye and GluA2 puncta in both type I and type II hair cells). These effects were partially reverted after the washout period. By RT-PCR, the most relevant changes in the vestibular epithelium were a decrease in Ctbp2 (ribeye) expression during the treatment which persisted into washout, and a decrease of Tnc (tenascin-C) during treatment with a full recovery after washout. In the vestibular ganglion, there was an increase in Gria2 (GluA2) and DLG4 (PSD95) during the washout period only.

In conclusion, adhesion and synaptic modifications are early events preceding hair cell extrusion during chronic ototoxicity in the mouse.

2) In vitro and in vivo neuroprotective evidences for new Imidazoline I2 receptor ligands.

Griñán-Ferré, C.[1] , Abás, S.[2] , Vasilopoulou, F.[1] ,Erdozain, A. M.,[3] Keller, B.,[4] Rodríguez-Arévalo, S.[2], Callado, L. F.[3], García-Sevilla, J.A.[4], Escolano, C.[2], Pallàs M[1].

[1]Secció de Farmacología. Institut de Neurociència. [2]Laboratory of Medicinal Chemistry (Associated Unit to CSIC). Facultat de Farmàcia i Ciències de l'Alimentació. Universitat de Barcelona, Av. Joan XXIII, 27-31, 08028 Barcelona, Spain. [3]Department of Pharmacology, University of the Basque Country, UPV/EHU, E-48940 Leioa, Bizkaia, Spain. Centro de Investigacion Biomédica en Red de Salud Mental, CIBERSAM. [4]Laboratory of Neuropharmacology, IUNICS/IdISPa, University of the Balearic Islands (UIB), Cra. Valldemossa km 7.5, E-07122 Palma de Mallorca, Spain.

Cognitive impairment associated with age is characterized by changes in neurons such as a reduction in the dendrite arborization and dendritic length, jointly with changes in a number of cell membrane receptors. Some of these receptors have a prominent role in brain functions as they are binding sites for the neurotransmitters modulating neurons' responses. Recently, imidazoline I2 receptors (I2-IRs) have been suggested as a potential therapeutic target for several CNS disorders such as Alzheimer's disease (AD).

We have recently reported the pharmacological characterization of a new family of (2-imidazolin-4-yl)phosphonates, prepared by a multicomponent microwaved-assisted reaction that fulfil the principles of green chemistry. Radioligand binding studies showed that they displayed an outstanding affinity for I2-IRs. Moreover, they showed also neuroprotective effects.

In the present work, we present further in vitro and in vivo studies on the neuroprotective properties of these compounds. To this end, after the positive results of the neuroprotection effects induced by Imidazoline I2 receptor ligands in primary neurons cultures, 12 months-old SAMP8 female mice were distributed in three groups: Control (n=10), compound 1 (n=8), and compound 2 (n=8). Mice were treated for 4 weeks with compounds 1 and 2 at the dose of 5 mg/kg/day added to drinking water.

Both compounds induced a significant increase in motor activity, measured by open field, and cognitive improvement, through novel object test. These changes were accompanied by a decrease in Tau hyperphosphorylation and amyloid precursor protein (APP) processing. A significant Bax and Calpain protein level diminution, and Bcl-2 increase, indicated modifications in apoptosis process. Finally, qPCR measured gene expression of IL-6, IL-1β and Cxcl10 inflammation markers as well as the oxidative stress markers Tnf-α, iNOS, Hmox1 and Aldh2 were reduced in the treated mice compared to the control groups. Altogether these results demonstrated the neuroprotective effect of these novel I2-IRs ligands in a neurodegenerative disease animal model of AD.

3) Molecular mechanisms involved in alterated mitochondria dynamics and function in Huntington's Disease.

Laura López-Molina [1,2,3], Marta Cherubini [1, 2, 3], Silvia Ginés [1, 2, 3].

[1]Departament de Biomedicina, Facultat de Medicina. Institut de Neurociències. Universitat de Barcelona, Barcelona, Spain. [2]Institut d'Investigacions Biomèdiques August Pi i Sunyer (IDIBAPS), Barcelona, Spain.3CIBERNED, Madrid, Spain.

Huntington's Disease (HD) is a neurodegenerative disorder characterized by the selective loss of striatal medium spiny neurons. Although the molecular mechanisms of this striatal vulnerability remain unclear, compelling evidences have point out that mitochondrial dysfunction could play a major role. Mitochondria are essential organelles for neuronal survival since they are the source of intracellular ATP and regulate Ca2+ homeostasis. For ensuring the maintenance of their function, mitochondria morphology and a balance between fusion and fission are strictly controlled.

Interestingly, defects of mitochondria dynamics processes have been found in HD, where mutant huntingtin enhances fission protein Drp1 activity. In the current work, we demonstrate that the increase of mitochondria fragmentation is accompanied by an unusual distribution of these organelles far from the endoplasmic reticulum (ER). Contact sites between both organelles called MAMs (mitochondria associated membranes) enables calcium effluxes from the ER to the mitochondria via VDAC-IP3R3 complex, reinforced by Grp75 and Mfn2 proteins. Biochemical analysis revealed specific changes of MAM-resident Ca2+ regulatory proteins in the striatum of knock-in HD mutant mice and human brain and disruption of ER-mitochondria contact sites in striatal cells under the presence of mutant huntingtin.

On the other hand, pharmacological Drp1 inhibition has proved to restore mitochondrial fragmentation and recuperate mitochondrial Ca2+ handling and crosstalk between ER and mitochondria. Taken together, these data support the hypothesis that alterations in mitochondrial dynamics may contribute to a major susceptibility of the striatum to huntingtin toxicity.

4) Associations between Single Nucleotide Polymorphisms in the mTOR pathway with early onset and severity of L-dopa induced dyskinesia in Parkinson's disease patients.

Núria Martín-Flores [1, 2 *], Rubén Fernández-Santiago [3,4,5 *], Francesa Antonelli [4], Catalina Cerquera [4], Verónica Moreno [4], Maria Josep Martí [3,4,5 $], Mario Ezquerra [3,4,5 $] and Cristina Malagelada [1,2 $].

[1]Department of Biomedicine, Unit of Biochemistry, Faculty of Medicine, Universitat de Barcelona, Barcelona, 08036 Catalonia (Spain). [2]Institut de Neurosciències, Universitat de Barcelona, Barcelona, 08036 Catalonia (Spain). [3]IDIBAPS-Institut d'Investigacions Biomèdiques August Pi i Sunyer, Barcelona, 08036 Catalonia (Spain). [4]Neurology Service, Hospital Clínic de Barcelona, Barcelona, Spain. [5]Centro de Investigación Biomédica en Red sobre Enfermedades Neurodegenerativas (CIBERNED).

[*] These authors have contributed equally to this work.

[$] These senior authors have contributed equally to this work.

Parkinson's disease (PD) is the second most common neurodegenerative disorder and is characterized by the degeneration of certain neuronal populations. The most effective drug to treat PD is levodopa (L-DOPA), the precursor of dopamine.

However, chronic treatment with L-DOPA triggers other motor complications. L-DOPA-induced dyskinesia (LID) is one of the most disabling problems for PD patients. Currently there is no method to predict which PD patient will develop LID. One strategy to identify possible molecular markers to predict L-DOPA treatment outcome involves the screening of single-nucleotide polymorphisms (SNP) in crucial signaling pathways associated to PD pathogenesis. mTOR signaling is one of these pathways.

For this reason, here we investigated potential associations between genetic variations in the genes of the mTOR pathway and the onset and severity of LIDs in patients with PD.

We selected 64 SNPs from 57 genes in the mTOR pathway. The whole cohort of study consisted in 1,819 subjects including 898 PD cases and 921 unrelated healthy controls. A total of 401 of the 898 PD cases had complete L-DOPA treatment and LID data registered in their clinical histories.

Here, we found new associations between the early appearance and the severity of LIDs with SNPs in components of the mTOR pathway. We have detected both single SNPs associations and epistatic interactions of SNPs that could predict the appearance or severity of LIDs.

The results in this study will help to design diagnostics tests to prevent/delay LID appearance and therefore, to improve PD patients' quality of life.

5) Corpora amylacea of human brain contain neo-epitopes that are recognized by natural IgM antibodies.

E. Augé [1,2], I. Cabezón [1,2], C. Pelegrí [1,2], J. Vilaplana [1,2].

[1] Facultat de Farmàcia i Ciències de l'Alimentació, Universitat de Barcelona, Barcelona. [2] Institut de Neurociències, Universitat de Barcelona, Barcelona

Corpora amylacea are structures of unknown origin and function that appear with age in human brains and are profuse in selected brain areas in several neurodegenerative conditions. They are constituted of glucose polymers and may contain waste elements derived from different cell types. Human brain sections from AD and elderly donors were analysed by immunohistochemistry. As we previously found on particular polyglucosan bodies in mouse brain, we report here that corpora amylacea present some neo-epitopes that can be recognized by natural antibodies, a certain kind of antibodies that are involved in tissue homeostasis. We also detected that these natural antibodies are frequently present as contaminants in many commercial antibodies and are responsible of high number of false positive immunostaining. We hypothesize that corpora amylacea, and probably some other polyglucosan bodies, are waste containers in which deleterious or residual products are isolated to be later eliminated through the action of the innate immune system. In any case, the presence of neo-epitopes on these structures and the existence of natural antibodies directed against them could become a new focal point for the study of both age-related and degenerative brain processes.

6) Dysfunctional inhibitory network dynamics in a Huntington's disease model revealed through large scale calcium imaging.

S. Fernández-García [1,2,3,4], M. Masana [1,2,3,4], J. G. Orlandi [1,2,3,4,5,6], G. García-Díaz Barriga [1,2,3,4], J. Soriano [5], J. Alberch [1,2,3,4].

[1] Universitat de Barcelona, Departament de Biomedicina, Facultat de Medicina - Barcelona, Spain. [2]Universitat de Barcelona, Institut de Neurociències, Barcelona, Spain. [3] Institut d'Investigacions Biomèdiques August Pi i Sunyer (IDIBAPS), Barcelona, Spain. [4]Centro de Investigación Biomédica en Red sobre Enfermedades Neurodegenerativas (CIBERNED), Madrid, Spain. [5]Universitat de Barcelona, Departament de Física de la Matèria Condensada, Facultat de Física, Barcelona, Spain. [6]University of Calgary, Department of Physics and Astronomy, Calgary, Alberta, Canada.

Striatum, the main hub of the basal ganglia circuitry, is the most affected region in Huntington's disease (HD). In HD, mutant-huntingtin (mHtt) causes an excitatory-inhibitory imbalance of the basal ganglia output pathways and induces motor symptoms. Although alterations of striatal Medium-sized Spiny Neurons (MSN) occur at early stages of the disease, little is known about how this translates into functional changes in the network dynamics. Using large scale calcium imaging, we have recorded simultaneously hundreds of cells from striatal primary cultures in WT and the R6/1 mouse model of HD and characterized the spontaneous activity patterns of every cell as well as their collective activity.

At single cell level, we identified three populations based on their fluorescence activity traces in both WT and R6/1. Type 1 (fast increase/exponential decay) characteristic of neurons; Type 2 (slow calcium transients) resembling astrocytic calcium waves; and Type 3 (non-detectable fluorescence changes). This heterogeneous cell population was confirmed by immunocytochemistry against neuronal and glial (MAP2/GFAP) markers. At the population level, both WT and R6/1 include a subset of neurons that display highly coherent activity, indicating the presence of a functional network. In basal conditions, R6/1 striatal cultures show increased bursting activity respect to WT. This difference is lost when the cultures are disinhibited through the blockade of GABAA receptors. This result indicates that striatal network dysfunction may arise from local disinhibition.

Further analysis is required to characterize the contribution of different neurotransmitter systems to the striatal network dynamics. Understanding functional network alterations in HD is fundamental to decipher initial key mechanisms to finally target early symptoms.

Posters presentations: Neurobiology and Neuropharmacology

7) Role of membrane raft microdomains in axonal repulsion to Netrin-1 and axonal growth.

Roselló C [1,2,3], Martínez-Mármol R [4], Hernaiz M [1,2,3] and Soriano E [1,2,3].

[1] Developmental Neurobiology and Neuronal Regeneration Lab. Department of Cell Biology, Physiology and immunology, University of Barcelona, Spain. [2] Centro de investigaciones en Red sobre Enfermedades Neurodegenerativas (CIBERNED), Madrid, Spain. [3] Vall d'Hebron Institut de Recerca (VHIR), Barcelona, Spain. [4] Queensland Brain Institute, Queensland University, Brisbane, Australia.

Axonal guidance during development of the nervous system is thought to be highly regulated through interactions of axons with attractive, repulsive, and trophic cues. Similar mechanisms regulate axonal regeneration after injury.

Netrin-1 is a diffusive guidance molecule that has been shown to influence the guidance of several classes of developing axons. Netrin-1 has been implicated as axonal guidance cue in the developing of both central and peripheral nervous system and recently it has emerged as an important chemo repellent after spinal cord lesion. Netrin-1 receptors are transmembrane associated proteins that can work either promoting or inhibiting the growth of axons towards a source of Netrin-1. Interestingly, some of Netrin-1 receptors can work modulating axonal growth in the presence of Netrin-1, or initiating neuronal death when Netrin-1 is absent. DCC, UNC5 and Neogenin are distributed along membranes into specialized microdomains termed as lipid rafts. Alteration of membrane lipid composition can modify the activity of raft-embedded proteins. To date, the role of DCC as axonal guidance and the role of DCC and UNC5 proteins as cell death depend on raft integrity. However, to our knowledge, no information has been revealed about the function of UNC5 proteins as mediators of Netrin-1-dependent axonal repulsion. Here we evaluate the raft-dependent repulsive role of Netrin-1 in a neuronal systems where Netrin-1 repulsion is crucial for the correct positioning of axonal projections, the cerebellar granular cells (EGL). Moreover, we also demonstrate how disrupting lipid rafts increases the number of filopodia and the growth cone area in CNS and PNS.

8) Role of Reelin in adult brain plasticity.

Vílchez Acosta A.[1,2]; Manso Y.[1,2]; Masachs N.[1,2]; Bosch C.[1,2,3]; Delgado-García JM.[4]; Gruart A.[4] and soriano E.[1,2,3].

[1] Developmental Neurobiology and Neuronal Regeneration Lab. Department of Cell Biology, Physiology and immunology, University of Barcelona, Spain. [2] Centro de investigaciones en Red sobre Enfermedades Neurodegenerativas (CIBERNED), Madrid, Spain. [3] Vall d'Hebron Institut de Recerca (VHIR), Barcelona, Spain. [4] Division of Neurosciences, Pablo de Olavide University, Seville, Spain.

Reelin is a large extracellular matrix glycoprotein with a crucial role in migration and positioning of cortical neurons during development in the mammalian neocortex. At embryonic stages, Reelin is expressed by Cajal-Retzius cells in the developing cortex. In this region, postmitotic neurons migrate in an ordered sequence that determines the normal "inside-out" pattern of layer formation. The mispositioning of cortical neurons, resulting from abnormal migration, causes severe layering malformations with functional consequences related with neurodevelopmental diseases such as schizophrenia, autism and epilepsy. In this context, one of the most studied phenotypes is the reeler mouse, which is caused by an autosomal mutation in the reelin gene. However the study of the effects of Reelin signalling in the adult brain is difficult in the reeler mouse model due to the marked developmental defects seen in this mutant. In the adult brain Reelin is mainly expressed by GABAergic interneurons of the cortex and hippocampus where presumably modulates developmental-reminiscent mechanisms that remain active throughout life. To unravel the function of Reelin in early postnatal and adult development we are characterizing different conditional floxed-Reelin KO mouse lines. Our results suggest that the absence of Reelin during adulthood has a profound impact on the structural and physiological plasticity of the cerebral cortex. . Furthermore, Reelin deficiency leads to several cognitive and behavioural deficits. Taken together our results suggest a causal relation between the absence of Reelin and structural and functional alterations in the hippocampus and cortex, which may be linked to disease pathogenesis.

9) 11β-HSD1 inhibition improved cognitive alteration modifying epigenetic marks in SAMP8 mice chronic mild stress exposure.

Dolors Puigoriol [1], Christian Griñán [1], Júlia Companys [1], Rosana Leiva [2], Eduard Otero [1], Santi Vázquez [2] and Mercè Pallàs [1].

[1] Unitat de Farmacologia i Farmacognòsia, Facultat de Farmàcia i Institut de Neurociències. Universitat de Barcelona, Av. Joan XXIII, 27-31, 08028 Barcelona, Spain . [2] Laboratori de Química Farmacèutica (Unitat Associada al CSIC), Facultat de Farmàcia i Institut de Biomedicina (IBUB), Universitat de Barcelona, Av. Joan XXIII, 27-31, 08028 Barcelona, Spain.

A key factor in age-related cognitive decline is elevated glucocorticoids (GCs) exposure. In fact, high levels of GCs have been found in elderly who exhibit learning and memory impairments and correlates with greater hippocampal atrophy. Moreover, there is evidence that aged mice with cognitive deficits show increased 11β-Hydroxysteroid dehydrogenase (11β-HSD1) expression and that overexpression of 11β-HSD1 displays a similar premature memory decline. Epigenetic takes part in control of transcriptional mechanisms one of the earliest emerging fields in senescence processes, playing a pivotal role in nuclear integration of intracellular signalling events to promote structural and functional adaptations underlying oxidative stress, inflammation and neuronal plasticity. The relationship between stress response and epigenetics in the brain is bidirectional and potentially reversible. Therefore, understanding the epigenetic modifications by which stressful environmental interacts in brain and affect learning and memory is essential in order to develop novel therapeutics to age-related pathologies and neurodegenerative diseases.

In the current study, we investigate the relationship between aging, chronic mild stress, behavioural abilities and cognitive impairment, as well as correlations with epigenetic alterations in SAMP8. Moreover, we assessed the ability of the novel pyrrolidine-based 11β-HSD1 inhibitor in 6 months-old female SAMP8 in which unpredictable chronic mild stress (UCMS) paradigm was applied for 3 weeks. 11β-HSD1 inhibitor (21mpk) was administered in control and UCMS mice in drinking water. Changes in behaviour and cognition were found in SAMP8 stressed mice compared to SAMP8 non-stressed through EPM or OF and NORT, respectively. These changes were accompanied by inflammatory and oxidative stress markers reduction. In addition, we found changes on epigenetic machinery and their epigenetic marks such as DNA methylation (5-mC) and histone acetylation levels. In conclusion 11β-HSD1 inhibitor treatment is revealed as a possible treatment of neurodegenerative diseases.

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10) Differential pattern of Tau phosphorylation in distinct hippocampal neuronal population in control and pathological conditions.

Eva Dávila [*1,2,3,4], Georgina Targa [*1,2,3,4], Jesús Ávila [2,5], Eduardo Soriano [1,2,3,4], Marta Pascual [1,2,3,4].

[1] Department of Cell Biology, University of Barcelona, Barcelona, Spain. [2] Centro de Investigación Biomédica en Red sobre Enfermedades Neurodegenerativas (CIBERNED, ISCIII), Spain. [3] Institut de Neurociències University of Barcelona, Spain. [4] Vall d'Hebron Institute of Research, Barcelona, Spain. [5] Centro de Biología Molecular Severo Ochoa (CSIC-UAM), Neurobiology Laboratory, Madrid Spain.

* Co-first author

Alzheimer's disease (AD) is characterized by the accumulation of β-amyloid peptide (Aβ) and hyperphosphorylated Tau protein. Both play a decisive role in synaptic and neuronal degeneration, which finally leads to cognitive impairment. We have recently reported the accumulation of phosphorylated Tau protein (P-Tau) at Thr231 in pyramidal cells, mossy cells, and Parvalbumin (PV)-positive hippocampal interneurons in VLW mice, which overexpress mutated human Tau. We have also described the absence of Thr231 P-Tau in the hippocampi of control mice. These data indicate a specific pattern of Tau phosphorylation in distinct subpopulations of hippocampal neurons in a mouse model of AD.

To determine the distribution pattern of P-Tau, we performed immunohistochemistry to detect Tau phosphorylation at Ser262 and Thr205 in hippocampal sections from control animals, VLW mice accumulating P-Tau, and J20 mice, which accumulate Aβ. Our first results demonstrate that P-Tau at Ser262 and Thr205 is present specifically in the soma of some hippocampal interneurons in WT mice, thereby pointing to a specific role of the phosphorylated protein at Ser262 and Thr205 in the soma of hippocampal interneurons in non-pathological conditions.

The analysis of VLW and J20 mice revealed that the density of hippocampal interneurons accumulating P-Tau at Ser262 and Thr205 is lower in VLW mice than in controls. Moreover, our data indicate an increase in the density of hippocampal interneurons accumulating P-Tau at Ser262 and Thr205 in J20 mice. This observation suggests that Tau hyperphosphorylation is induced by the presence of Aβ. Hippocampal interneurons form a heterogeneous group of neurons. Three main types can be determined on the basis of their content of PV, Calbindin (CB) or Calretinin (CR). By double immunodetections, we demonstrate that P-Tau at Ser262 is present in the soma of some hippocampal interneurons containing PV, CB or CR in control, VLW, and J20 mice. In contrast, only some PV- and CB-positive interneurons of the three animal models accumulate P-Tau at Thr205 in the hippocampus.

All these data reveal a differential phosphorylation pattern of Tau protein in hippocampal interneurons in control and pathological conditions and point to a novel specific function of Tau in the soma of this cell population.

11) A central role of NCAM2 in neuronal polarization and differentiation.

Parcerisas A. [1,2,4,5], Ortega A. [1,2,4,5], Viais R. [6], Figueiro-Silva J. [3], Trullas R. [3], Lüders .J [6], Pujadas L. [1,2,4,5] and Soriano E. [1,2,4,5].

[1] Developmental Neurobiology and Regeneration Lab. Department of Cell Biology, University of Barcelona, Barcelona. [2] Centro de Investigación Biomédica en Red sobre Enfermedades Neurodegenera8vas (CIBERNED, ISCIII). [3] Neurobiology Unit, Institut d'Investigacions Biomèdiques de Barcelona, Consejo Superior de Investigaciones Científicas, Institut d'Investigacions Biomèdiques August Pi i Sunyer, Barcelona. [4] Vall d´Hebron Institut de Recerca (VHIR), Barcelona. [5] Institut de Neurociències, Barcelona. [6] Institue for Research in Biomedicine Barcelona

The proper neuronal polarity and long-term maintenance of neurons is crucial for correct brain functioning, with special relevance to the connectivity orchestration during development and learning processes. Furthermore, changes in the neuronal polarity are one of the keys features in neurodegenerative diseases.

Cell Adhesion Molecules (CAMs) play an important role during brain development and brain plasticity processes, being involved in neurite outgrowth, synaptogenesis and synaptic plasticity. Neural cell adhesion molecule 2 (Ncam2) is a CAM family member homologous to Ncam1 with a similar extracellular domain. NCAM2 expression pattern in brain indicates a putative important role of this protein in both embryonic and adult brain functioning.

Ncam2 has been extensively studied in the olfactory system where it plays an important role in axonal fasciculation and neurite outgrowth. Recently, different studies have revealed that Ncam2 also participates in the formation of filopodia and neurite outgrowth in cortical neurons. Nevertheless its function in hippocampal development remains largely unknown.

We used different cell biology and molecular approaches, including loss-of- and gain-of-function models, to determine the role of Ncam2 in brain development. Our data show that Ncam2 is expressed in the hippocampus and that it is essential for appropriate neuronal polarity. Ncam2 plays a role in neurite outgrowth, axonal branching, and dendrite formation. The proteomic approach performed show that Ncam2 interacts with more than a 100 proteins including cytoskeleton components and regulatory molecules. Preliminary experiments indicate that Ncam2 levels modify different cytoskeletal components, associated to the microtubular network. Taken together, our results highlight Ncam2 as an important molecule for the development, specification and connectivity of the hippocampal formation.

12) Dexibuprofen prevents cognitive and metabolic parameters associated with Alzheimer disease in APPswe/PS1dE9 mice.

Miren Ettcheto [1,2,3,4], Elena Sánchez-López [5,6], Sergi Gorriz [1], Oriol Busquets [1,2,3,4], Carme Auladell [4,7], Jaume Folch [2,3], Antoni Camins [1,2,3,4].

[1] Departament de Farmacología, Toxicología I Química Terapéutica, Facultat de Farmàcia i Ciències de l'Alimentació, Universitat de Barcelona, Barcelona, Spain. [2] Departament de Bioquímica, Facultat de Medicina i Ciències de la Salut, Universitat Rovira i Virgili, Reus, Spain. [3] Biomedical Research Networking Center in Neurodegenerative Diseases (CIBERNED), Madrid, Spain. [4] Institut de Neurociències, Universitat de Barcelona, Barcelona, Spain. [5] Unitat de Farmacia, Tecnologia Farmacèutica i Fisico-química, Facultat de Farmàcia, Universitat de Barcelona, Barcelona, Spain. [6] Institute of Nanoscience and Nanotechnology (IN2UB), University of Barcelona, Barcelona, Spain. [7] Departament de Biologia Cel·lular, Fisiologia i Immunologia, Facultat de Biologia, Universitat de Barcelona, Barcelona, Spain.

Alzheimer´s disease (AD) is the most common form of dementia. Although its etiology remains unclear, multiples studies have demonstrated its link to metabolic disorders such as diabetes tipus II. Nowadays, there are no effective treatments to AD, however, the chronic consumption of non-steroidal anti-inflammatory drugs have been described that could reduce the risk of this disease. Thus, the main objective of this study was to evaluate the effects of dexibuprofen (DXI), the active enantiomer of ibuprofen, in 6 month-old APPswe/PS1dE9 (APP/PS1) female mice fed with high fat diet (HFD).

DXI was orally administrated at a dose of 20 mg·kg-1·d-1 for 3 months until their sacrifice. As expected, mice fed with HFD showed a significant increase of their weight compared to mice fed with control diet in both, treated and untreated animals; however, APP/PS1 HFD DXI mice weighted significantly less than APP/PS1. Besides, DXI reverted the negative metabolic parameters produced by HFD measured by glucose and insulin tolerance test. Moreover, we demonstrated that DXI decreased the glial reactivity, the number and size of beta-amyloid plaques and improved cognitive decline involved in AD.

In conclusion, our findings demonstrated that in a model of familial AD exacerbated with HFD, DXI could be an effective drug for the prevention of the disease together with others.

13) Effect of Resveratrol on epigenetic inheritance improves cognitive impairment in female SAMP8 by reducing oxidative stress and neuroinflammation markers.

Vanesa Izquierdo Cadenas, Verónica Palomera Ávalos, Christian Griñan Ferre y Mercè Pallàs Lliberia

Alzheimer disease (AD) is characterized by its multiple complex interaction between genetics and non-genetic risk such as environmental and lifestyle factors. Epigenetic mechanisms regulate changes in gene expression without altering the DNA information. Moreover, these epigenetic marks pass phenotypic change to subsequent generations through different mechanisms, the process is called Epigenetic inheritance. Nutrition mediates epigenetic changes that can lead to improve health outcomes. Resveratrol is a polyphenol with antioxidant and anti-inflammatory effects. Moreover, mediates epigenetic changes involved in the function of the Central Nervous System (CNS).

In the present work we study the epigenetic changes of dietary resveratrol in female AD mice model (SAMP8) through generations. Differences in gene expression of DNA methyltransferases Dnmt3a, Dnmt3b and Ten-eleven translocase Tet1 that regulate respectively, methylation (5-mC) and hydroxymethylation (5-hmC) of DNA and G9a methyltransferase involved in histone methylation modifications, were found.

Importantly, it has been demonstrated that epigenetic changes produced by resveratrol in SAMP8 were maintained across generations.

In addition, we studied transcription factor Nf-κβ and its target genes related to neuroinflammation such as Il-6, Cxcl10 and Tnf-α. Also, we found differences in the gene expression of the transcriptional factor Nrf2 and its target genes related to oxidative stress such as Hmox1, Aox1 and Cox2.

Taken together, our results demonstrated a neuroprotective effects by resveratrol through epigenetic changes, transmitted across three generations. It can be suggested that a complex set of factors, including nutrition, can be a key factor in epigenetic inheritance leading to a healthy penothype.

14) The absence of JNK1 or JNK3 reduces neurogenesis in the subgranular zone of the hippocampus in mice treated with kainic acid.

Landa J., Caruana P., Martinez M., Steele L., Castro-Torres R.D., Verdaguer E. Auladell C.

Department of Cell Biology, Phisiology and Immunology, University of Barcelona.

The c-Jun N-terminal kinases (JNKs) are members of the MAPK (mitogen-activated protein kinase) family and can be activated in response to various stimuli, such as growth factors, inflammatory cytokines and neurotoxins. These kinases are essential for the correct development of the mammal brain.

There are multiple splice variants of JNKs, encoded by three different genes: JNK1, JNK2 and JNK3. Previous studies demonstrated that the absence of JNK3 isoforms prevents the damage caused by kainic acid (KA) in mice, a non-degradable structural analogue of glutamate that binds to its receptor, followed by uncontrolled calcium entrance into neurons. This model reproduces some features of the temporal lobe epilepsy (TLE).

The aim of this project is to analyze the role of JNK isoforms in adult hippocampal neurogenesis, specifically, in the subgranular zone of the dentate gyrus (SGZ). In order to carry out this proposal, different subtypes of neural stem cells (NSC) of the SGZ were characterized in the wild type (WT) and in knockout mice (KO) (jnk1-/-, jnk2-/- and jnk3-/-) at basal conditions and following intraperitoneal injections of KA. The results showed no differences between WT and KO mice neither in the early progenitors nor in amplified neurons at basal conditions, although an increase of immature neurons was identified in jnk1-/- mice. However, the different NSC subpopulations were not increased in jnk1-/- and jnk3-/- mice after the KA treatment, contrary to WT and jnk2-/-, where they did increase.

This data supported that the neuroprotection of jnk1-/- and jnk3-/- can be reported through the reduction of neurogenesis, evidencing that both isoforms could be potential targets to prevent the brain damage developed after induction of excitability.

Posters presentations: Cognitive Neuroscience and Neuropsychology

15) Subcortical sound processing modulates simple auditory perceptual decisions

Natàlia Gorina-Careta and Carles Escera;

Brainlab-Cognitive Neuroscience Research Group, Department of Clinical Psychology and Psychobiology, University of Barcelona.

Simple auditory perceptual decisions are thought to be mediated by neural computations and processes occurring in the ventral auditory pathway. Indeed, it has been shown that throughout the structures that conform this pathway, auditory information is organized and processed in a hierarchical manner, with complex feature processing taking place in higher areas of the auditory hierarchy. However, recent studies have demonstrated that sound processing is not carried out exclusively in cortical regions so that the subcortical auditory pathway also has an active role in the perception and processing of the incoming sounds. These findings are consistent with the hypothesis of a distributed network for perceptual organization. To assess how auditory simple cortical perceptual processing relies on preceding subcortical processing, here we recorded the Frequency-Following Response (FFRs) to a set of pure tones of 20 different frequencies ranging from 121 to 577 Hz, as well as the behavioral response times to these same sounds before and after the FFR recording. Together, our findings support that subcortical sound processing has an important role in making of simple perceptual decisions, thus providing evidence that the evidence accumulation models are not only limited to cortical activity as they should also take into consideration the subcortical contribution.

16) The age of onset of substance use influences the neuropsychological performance of polyconsumer men under treatment.

M. M. Capella [1], L. Río [1,2] & A. Adan [1,2].

[1] Department of Clinical Psychology and Psychobiology, University of Barcelona, Barcelona, Spain. [2] Institute of Neurosciences, University of Barcelona, Barcelona, Spain)

Background: Current models of development and maintenance of Substance Use Disorders (SUD) highlight the role of biological and neurocognitive factors stands, in addition to environmental factors.

Objective: In this study, we evaluate the influence of age of onset of substance use (OSU), considered at 16 years or before (OSU≤16) and at 17 years or later (OSU≥17), on the clinical course and neuropsychological performance of SUD patients. In addition, we also explore the effect of years of education, duration of drug use and premorbid intelligence quotient (IQ) on the cognitive results obtained.

Methods: 80 male polyconsumers, 41 with OSU≤16 and 39 with ICS≥17, were assessed though an exhaustive neuropsychological battery. The patients were under treatment with at least 4 months of abstinence confirmed by urinalysis.

Results: OSU≤16 group presented a worse clinical state, as well as a lower premorbid IQ and worse performance in processing speed, visual perception and planning skills ($p<0.045$; $\eta p2>0.06$, in all cases). The duration of drug use may account for the differences in planning and processing speed. ($p=0.0001$, in both cases).

Conclusions: In patients with OSU≤16, the lower performance in the verbal subcomponent of the IQ could be a premorbid characteristic, while the deficits in this performance subcomponent and in the visuoperceptual skills could be acquired. Take into account the duration of consumption in the evaluation and intervention of addiction can provide new elements that improve the approach and rehabilitation of SUD patients, although future research is needed to clarify these issues.

17) Olfactory loss in Parkinson's disease: a 4-year follow-up study.

Anna Campabadal, [1,2]; Barbara Segura, [1]; Hugo C. Baggio, [1]; Carme Uribe, [1]; Alexandra Abos, [1]; Anna Isabel Garcia-Diaz, [1]; Maria Jose Marti, [3,4]; Francesc Valldeoriola, [3,4]; Yaroslau Compta, [3,4]; Nuria Bargallo, [5]; Carme Junque, [1,2,3].

[1]Medical Psychology Unit, Department of Medicine. Institute of Neuroscience, University of Barcelona. Barcelona, Catalonia, Spain. [2]Institute of Biomedical Research August Pi i Sunyer (IDIBAPS). Barcelona, Catalonia, Spain. [3]Centro de Investigación Biomédica en Red sobre Enfermedades Neurodegenerativas (CIBERNED), Hospital Clínic de Barcelona. Barcelona, Spain. [4]Movement Disorders Unit, Neurology Service, Hospital Clínic de Barcelona. Institute of Neuroscience, University of Barcelona, Barcelona, Catalonia, Spain. [5]Centre de Diagnòstic per la Imatge, Hospital Clínic, Barcelona, Catalonia, Spain.

Background: Olfactory dysfunction is present in a large proportion of patients with Parkinson's disease (PD) upon diagnosis. However, its progression over time has been poorly investigated. The few available longitudinal studies lack control groups or MRI data.

Objective: To investigate the olfactory changes and their structural correlates in non-demented PD over a four-year follow-up.

Methods: We assessed olfactory function in a sample of 25 PD patients and 24 normal controls of similar age using the University of Pennsylvania Smell Identification test (UPSIT). Structural magnetic resonance imaging data, obtained with a 3-Tesla Siemens Trio scanner, were analyzed using FreeSurfer software.

Results: Analysis of variance showed significant group (F=53.882; P<.001) and time (F=6.203; P=.016) effects, but the group-by-time interaction was not statistically significant. UPSIT performance declined ≥ 1.5 standard deviations in 5 controls and 7 patients. Change in UPSIT scores of patients correlated positively with volume change in the left putamen, right thalamus, and right caudate nucleus.

Conclusion: Olfactory loss over time in PD and controls is similar, but we have observed significant correlation between this loss and basal ganglia volumes only in patients.

18) Visual hallucinations are linked to increased cerebral atrophy and worse neuropsychological performance in non-demented Parkinson's disease patients.

Carme Uribe [1,2]; Carlos Segura-Escribano [1]; Barbara Segura [1,2]; Hugo C Baggio [1,2]; Anna Campabadal [1,2,3]; Alexandra Abos [1,2]; Anna I Garcia-Diaz [1,2]; Maria-Jose Marti [2,3,4,5]; Yaroslau Compta [2,3,4,5]; Francesc Valldeoriola [2,3,4,5]; Carme Junque [1,2,3,5].

[1] Department of Medicine, Faculty of Medicine, University of Barcelona, Barcelona, Catalonia, Spain. [2] Institute of Neurosciences, University of Barcelona, Barcelona, Catalonia. [3] Institute of Biomedical Research August Pi i Sunyer (IDIBAPS), Barcelona, Catalonia, Spain. [4] Movement Disorders Unit, Neurology Service, Hospital Clínic de Barcelona, Barcelona, Catalonia, Spain. [5] Centro de Investigación Biomédica en Red sobre Enfermedades Neurodegenerativas (CIBERNED), Hospital Clínic de Barcelona, Barcelona, Catalonia, Spain.

Background: The presence of visual hallucinations in Parkinson's disease (PD-VH) is a risk factor for rapid progression to dementia. However, characteristics of non-demented PD-VH are not completely understood. We aimed to investigate the neuropsychological characteristics and brain atrophy in limbic, paralimbic and neocortical regions in non-demented PD-VH patients.

Participants and Methods: T1-weighted magnetic resonance images, clinical and neuropsychological assessment were obtained in a group of 44 PD patients (22 with and 22 without visual hallucinations) and 22 healthy controls (HC) matched for sex, age and educational level. Cortical atrophy was assessed using a general linear model in FreeSurfer software whereas FSL-VBM ROIs approach was applied to test the involvement of a priori selected limbic regions including the amygdala, hippocampus and ventral striatum.

Results: PD-VH patients were more depressed and had more severe neuropsychiatric symptoms than HC and PD patients without VH. They also showed worse performance in total recall (p=0.016) and immediate recall (p=0.002) from Rey's Auditory-Verbal Learning Test, Symbol Digits Modalities Test oral version (p=0.002), Stroop Words (p=0.011) and Colours (p=0.010) than HC. Both PD groups showed cortical thinning in temporal, inferior parietal and occipital areas when compared to HC. Patients with VH also showed cortical thinning in posterior cingulate region when compared to HC. Regarding subcortical limbic regions, PD-VH showed significant reductions in right amygdala, bilateral posterior hippocampus and left ventral striatum when compared to PD without VH.

Conclusions: These results suggested that neurodegenerative process in non-demented PD-VH is mainly associated with brain atrophy in subcortical limbic regions.

19) Effect of motion-contaminated functional connectivity in movement disorder patients.

Alexandra Abos [1], Hugo C Baggio [1], Barbara Segura [1], Carme Uribe [1], Anna Campabadal [1,2], Anna Garcia-Diaz [1] and Carme Junque [1,2,3].

[1] Medical Psychology Unit. Department of Medicine, Faculty of Medicine, University of Barcelona, Barcelona, Catalonia, Spain. [2] Institute of Biomedical Research August Pi i Sunyer (IDIBAPS), Barcelona, Catalonia, Spain. [3] Centro de Investigación Biomédica en Red sobre Enfermedades Neurodegenerativas (CIBERNED), Hospital Clínic de Barcelona, Barcelona, Catalonia, Spain.

Background: Graph representation of a brain network, usually referred as connectome, can be characterized through resting-state functional magnetic resonance imaging (fMRI). A major issue concerning fMRI is susceptibility to motion, which can lead to abnormal connectivity values. Here we assess the effect of motion contaminated functional connectivity in movement disorder patients.

Methods: 164 subjects (62 healthy controls (HC), 57 Parkinson's disease (PD) patients, 29 Multiple system atrophy (MSA) patients and 16 Progressive supranuclear palsy (PSP) patients) with 3T resting-state fMRI data were included. From a 10 minutes fMRI sequence, the best 7 minutes (with less movement) and the worse 7 minutes (with more movement) were selected based on framewise displacement. Functional connectomes were created using the Brainnetome Atlas [Fan et al., 2016]. An in-house algorithm combining network-based statistics and threshold-free connectivity analysis was used to identify connections with group-level effects.

Results: Network-based statistics with threshold-free cluster enhancement detected 233 significant connections (familywise error-corrected $p<.05$) between HC and MSA patients and 420 significant connections (familywise error-corrected $p<.05$) between HC and PD patients, when using the best 7 minutes sequence. No significant connections where observed in the motion corrupted sequence. Mean connectivity values (both in the overall connectomes and in the connections where intergroup effects were found) were lower in the groups of patients than in the HC group. The effect of motion in the connectivity leads to an increase of connectivity values in the worse 7 minutes sequence in comparison with the best 7 minutes sequence.

Conclusions: These results confirm previous observations of the critical effect of movement in connectivity studies. More importantly, these findings demonstrate that, if possible, selecting the 7 less corrupted minutes of the sequence can help minimizing this problem.

20) The frequency following response (FFR) in newborns.

Teresa Ribas[1,2,3], Laura Almeida [3,4] , Jordi Costa-Faidella [1,2,3], Montse Plana [3,4], MJ Corral [1,2,3], M. Dolores Gómez-Roig [3,4], Carles Escera [1,2,3].

[1]Institute of Neurosciences, University of Barcelona (Spain). [2] Brainlab – Cognitive Neuroscience Research Group. Department of Clinical Psychology and Psychobiology, University of Barcelona (Spain). [3] Institut de Recerca Sant Joan de Déu, Esplugues de Llobregat (Barcelona, Spain). [4] BCNatal – Barcelona Center for Maternal Fetal and Neonatal Medicine (Hospital Sant Joan de Déu and Hospital Clínic), University of Barcelona, Barcelona (Spain).

The frequency-following response (FFR) is a non-invasive electrophysiological measurement of the encoding of the temporal and spectral characteristics of the evoking sound in a cortico-subcortical auditory network. Abnormal FFRs are found in children with auditory processing disorders, dyslexia and autism. Thus, the FFR may offers a neurophysiological marker of the efficient encoding of speech sounds related to literacy abilities. The FFR as a potential predictor of communication skills has been shown in school-aged children and in infants with ages between 3 and 10 months. Yet little is known about the neural transcription of speech sounds in newborns. The possibility to record FFR in newborns has been suggested in no more than ten studies so far, showing a remarkable similarity between the adult and the newborn responses. However, before the FFR can be used in the clinics, normative values need to be established.

The aim of this study is the establishment of a normative database depicting the standard variability found in different parameters extracted from the newborn FFR. The FFR was recorded from a sample of 50 neonates aged 12-144 hours after birth, to syllables /da/ and /ga/ (170 ms long; F0=113 Hz; intensity: 55 dB SPL, 2000 sweeps, delivered with alternating polarities). Newborns were included after passing the universal hearing screening. Four subjects were subsequently excluded because wave V could not be identified. The F0 signal-to-noise ratio (SNR) was strongly represented in most newborns (SNR /da/: mean=3.98, SD=2.44; SNR /ga/: mean=3.94, SD=2.10) and differed statistically between tokens ($t(45)=-3.49$, $p=0.001$), with two outliers (SNR<1.5). Higher harmonic components were not reliably present in all subjects. In the time domain, onset latencies were assessed (/da/: mean=10.06 ms, SD=1.42; /ga/: mean=9.70 ms, SD=1.01; $t(45)=3.084$, $p=0.003$), indicating that the stimulus with the highest second formant (/ga/) elicited a faster response than the stimulus with the mid-frequency formants (/da/), in agreement with previous studies. Other objective indices have been studied to evaluate the accuracy and strength of pitch processing. Our study confirms the feasibility of recording the FFR in newborns at the maternity unit from the very onset of life and provides normative data from a preliminary sample of 46 newborns. Detecting an abnormal FFR in a newborn could lead to an early intervention that, given the plasticity of the underlying neurophysiological machinery, could promote an improvement in the encoding of speech sounds and thus avoid cognitive impairment.

21) Physiological stress in overweight adults is related to cortical thinning of prefrontal areas.

Ottino, J.[1,3,7] ; García-García, I.[1,3,5]; Garolera, M.[2,4,9] ; Caldú, X.[1,3,7] ; Segura, B.[3,7,8] ; Prats, X.[1] ; Sender, M.J.[6] ; Jurado, M.A. [1,2,3,7].

[1] Departament de Psicologia Clínica i Psicobiologia, Universitat de Barcelona.[2] Grup Consolidat de Neuropsicologia. Universitat de Barcelona. [3] Institut de Neurociènces. Universitat de Barcelona. [4] Unitat de Neuropsicologia (Hospital de Terrassa). Consorci Sanitari de Terrassa.
[5] Montreal Neurological Institute, McGill University. [6] CAP Terrassa Nord. Consorci Sanitari de Terrassa.[7] Institut de Recerca Sant Joan de Déu.[8] Departament de Medicina. Universitat de Barcelona. [9] Brain, Cognition and Behavior Clinical Research Group. Consorci Sanitari de Terrassa.

Overweight (body mass index or BMI ≥ 25 kg/m2) and stress have both become a worldwide major problem given their links with cognitive decline and brain atrophy. The prefrontal cortex alterations are linked to adiposity and stress, however the synergic effects that both conditions have over PFC areas has not been addressed enough yet. Hence, the aim of this study was to assess the link between physiological chronic stress and brain integrity in healthy overweight adults through cortical thickness analysis of the inferior prefrontal gyri (IPG), a key region related to eating behavior. Forty-two healthy overweight-to-obese participants (17 males, mean age 32.07 ± 5.97, age range 21-40, BMI 32.24 kg/m2 ± 5.48) were recruited from the Consorci Sanitari de Terrassa and underwent magnetic resonance imaging in a 3T MAGNETOM Trio (Siemens, Germany) in the Hospital Clínic de Barcelona to obtain high resolution T1-weighted MPRAGE 3D scans. We calculated a chronic-stress index with sixteen biomarkers and a percentile-risk procedure (binary scores of "1" for biomarkers higher than percentile 75) using cut-off points from a normative lean sample with similar sociodemographic characteristics. Bilateral inferior prefrontal gyri (IPG) mean thickness was obtained through FreeSurfer software (v.5.3) automated cortex parcellation (Fig. 1). Partial correlations controlling for age and years of education, and for multiple comparisons as well ($p < 0.016$) were conducted between left and right IPG mean thickness and the chronic stress index in IBM SPSS Statistics (v.23.0). Higher chronic-stress indexes were related to a decrease in cortical thickness in the left IPG ($r= -0.434$, $p = 0.005$) (Fig. 2). Our work suggests that the presence of high levels of chronic stress in healthy overweight young adults are linked to decreases in gray matter in regions involved in regulating eating behavior.

Posters presentations: Cognition, Behaviour and Computation

22) Spatial distribution in semi-free ranging macaques: Macaca tonkeana and Macaca fascicularis

Laura Mármola[2], Hélène Meunier[2,3], Ruth Dolado[1,2], Francesc S. Beltrana[1]

[1]Institute of Neurosciences, University of Barcelona, Barcelona, Spain
[2]Centre de Primatologie, Université de Strasbourg, Niederhausbergen, France
[3]Laboratoire de Neurosciences Cognitives et Adaptatives, CNRS et Université de Strasbourg, Strasbourg, France

The spatial distribution (i.e., individuals' proximity and dominant's centrality) is a key factor to understand social structure in Macaca species and varies between the different grades of the genus. In grades 1 and 2 dominant individuals are in the center of the group, near to individuals of similar rank, while subordinates are in the periphery. In grades 3 and 4 dominant individuals do not occupy central positions in the group and the spatial distribution is not rank-dependent. We tested two groups of semi-free ranging M. fascicularis (grade 2; N=16 individuals) and M. tonkeana (grade 4; N=22 individuals) housed at the Primate Centre of Strasbourg University. We used the procedure of Dolado & Beltran (2011) to obtain the individuals' proximity, the group dominance rank was calculated using David's score (David, 1987) and we also calculated the centrality of dominants according to Hemelrijk (1998). The results showed in M. tonkeana close proximities among females, some juveniles and the dominant male, being the other adults males further away, but inside each subgroup the proximity was not determinate by the rank. In the M. fascicularis group the proximity between individuals was determined by the rank. Moreover, the dominant male was more central in M. fascicularis but not in M. tonkeana. Our results agreed with the spatial differences expected on different grades of the Macaca genus, being measured for first time these differences in semi-free ranging groups.

23) Exploring autobiographical memories for real life sequences of event episodes with a wearable camera

Berta Nicolás, Mariela Dimiccoli, Valeria Marzano, Petia Radeva, Lluís Fuentemilla.

Universitat de Barcelona (UB) Department of cognition, development and education Psychology. Department of Mathematics and Computer Science Universitat de Barcelona (UB). Institute of Neuroscience Universitat de Barcelona (UB).

A current hallmark in Autobiographical Memory (AM) research is to unravel how individual sequences of real-life event episodes are encoded and retrieved from long-term memory. To address this question experimentally, we recorded electroencephalographic activity (EEG) while participants retrieved their individual AMs cued by pictures taken automatically by a wearable camera from the past one-week daily life. As our experience is continuous, we sampled real life experiences into segments of context-based episode units identified automatically by a semantic regularized clustering algorithm (SRclustering) that groups together temporally adjacent images sharing contextual and semantic attributes (extracted employing a convolutional network based approach)This approach allowed a simplification and an unbiased identification of the possible underlying structure of the sequential nature of the autobiographical experience. Our preliminary results show that our AM sampling method was effective as it lead each participant to make accurate judgments of their memories. In addition, we also found picture cues evoked reliable neural responses (i.e., Event-Related Potentials ERPs) associated to successful memory recollection at individual level. We conclude our approach may provide opportunities to investigate the underlying structure of individual and real-life AMs.

24) Sequential bilingualism breaks essentialist reasoning about language

J. Birules[1], C.Saumell[1], L. Bosch[1], K. Byers-Heinlein[2], & F. Pons[1]

[1]Department of Cognition, Development and Educational Psychology, Universitat de Barcelona, Pg. Vall d'Hebron 171, 08035 Barcelona, Spain. [2]Department of Psychology, Concordia University, Canada.

Four to five-year-old monolingual children have been found to believe language is an innately determined and stable trait, whereas race could be transformable (Kinzler & Dautel, 2012). In a switched-by-birth paradigm, Byers-Heinlein & Garcia (2015) showed sequential bilingual children had reduced essentialist believes about language, animal vocalizations and animal physical traits. Following these studies, we investigated essentialist biases about language and race in Catalan Spanish four to six-year-old simultaneous and sequential bilingual children. Experiment one consisted of Kinzler and Dauter 2012 task, which juxtaposes race (black and white) and language (Catalan-German or Catalan-Spanish). In experiment two, children were presented with the switched-at-birth paradigm from Byers-Heinlein and Garcia, 2015. Results show Spanish monolingual and Spanish-Catalan simultaneous bilingual children did not favour language over race in the two language conditions, nor believed the traits came from the birth parents in experiment two. In contrast, sequential bilingual children were at chance level in experiment one, and believed all traits would come from the adoptive parents. These results indicate the learning experience of a sequential bilingual can reduce children's essentialist beliefs about not only language but also race and animal's physical traits, as if extrapolating this linguistic experience into a more general flexibility in essentialist reasoning.

25) Eye-Hand Coordination in Interception with Delayed Visual Feedback

Clara Cámara[1,2] , Cristina de la Malla[3] , Joan López-Moliner[1,2] & Eli Brenner[3]

[1] Institut de Neurociències, Universitat de Barcelona. [2] Facultat de Psicologia, Universitat de Barcelona. [3] Faculty of Behavioural & Movement Sciences, Vrije Universiteit Amsterdam

When performing many common manual tasks, people's eye movements appear to be well coordinated with the movements of their hands. People readily learn to intercept a moving target with a cursor even if the cursor is delayed with respect to the hand by about 200ms. With such a delay, the position of the cursor is dissociated from that of the hand. We here examine how such adaptation influences eye-hand coordination. We recorded eye movements while subjects tried to intercept targets that were moving in different directions at different velocities. The experiment had 4 phases that differed in the feedback that was provided about the on-going movement of the hand. In the first phase they had full vision of the hand. In the second phase they had no visual information about the hand's movement. In the third (adaptation) phase they saw a cursor representing the hand. The fourth phase was identical to the second. During the adaptation phase we increased the delay between the hand and the cursor by 1ms on each trial until the delay was 200ms. When subjects could see their hand they hit the target with their hand. When feedback was removed they intercepted the target's path about 100ms ahead of the target. When a cursor represented the hand, they hit the target with the cursor, despite the delay, so that for large delays the hand passed even further ahead of the target. When the cursor was removed the hand gradually returned to crossing the target's path about 100ms before the target. In all cases the eyes simply pursued the target. In the adaptation phase the eyes continued to follow the target after the hand had crossed its path, presumably to obtain accurate feedback about the outcome of the movement.

26) Moderate Influence of Object Size Variability on Visual Gravity Judgements

Björn Jörges, Lena Slupinski, Joan López-Moliner.

Institut de Neurosciències, Universitat de Barcelona

A strong earth gravity prior lends a significant contribution to many cognitive functions regarding perceptuo-motor skills like reaching, catching or throwing (Jörges & López-Moliner, 2017). But what about earth-discrepant gravities? How do humans perceive these visually? A previous experiment suggests that mainly zeo optic variables (namely the Visual Angle and the Elevation Angle) play a role in extracting the gravity from an observed parabolic trajectory, along with a representation of the object size. In the present experiment, we investigate how variability of object size influences visual gravity judgments. To this end, participants (n=10) were asked to judge in a two-alternative forced-choice task which of two parabolas had the higher underlying gravity. The comparison parabola could be governed by one of seven gravities (0.7 g – 1.3 g), while the reference parabola was always governed by 1g. Both could have one out of two initial vertical velocities (3.7 or 5.2 m/s) and one out of two horizontal velocities (6 or 8.3 m/s). Additionally, we manipulated variability of ball size (drawn from a Gaussian distribution with a mean of 0.033 m and a SD of 0.02 m or 0.005 m, respectively presented in blocks). Participants displayed a slight decrease in sensitivity in the high variability condition as well as a bias to judge smaller balls as having a higher gravity. We conclude that an adequate representation of the actual ball size has some beneficial effects on the extraction of gravity values underlying parabolic trajectories.

27) Variability in movement speed can predict temporal adaptation in an interception task

Elisabeth Knelange & Joan López-Moliner

Vision & Control of Action group

To be successful at intercepting moving objects we need to predict the spatiotemporal features of the motion of the object and of our hand. The errors we make can result in updates of these predictions for future interceptions. Although variability in task performance is often seen as an unwanted consequence of noise, recent studies claim that variability in baseline performance can help adapt to a perturbation task. This finding suggests that initial variability in performance can help explore the spatial demands of the task. To see if this relationship can also be found in interception, we studied the link between the variability of movement velocity during baseline trials, and the adaptation to a temporal perturbation. 20 subjects performed an interception task on a graphic tablet with a stylus pen. A target moved from left to right or right to left, with varying speed across trials. Participants were instructed to intercept this target with a straight shooting movement. Their movements were represented by a cursor on a screen above the tablet, which blocked the view of their hand. The first part of the cursor's trajectory was also blocked from view to prevent online corrections. After a baseline phase of 80 trials, a temporal delay of 100 ms was introduced to the cursor representing the hand. This delay initially caused the participants to produce temporal errors in the performance, but they quickly adapted to the delay of the cursor to account for these errors. We found that participants with higher variability in movement speed during the baseline phase show a better adaptation to a temporal delay in the adaptation phase. This finding suggests that high variability in movement velocity can provide people with tools to learn from temporal errors in interception.

Posters presentations: Neurology and Psychiatry

28) Role of TRESK channels in sensory neurons involved in non-histaminergic itch

Andrés, A.[1,2,3], Castellanos, A.[1,2,3], Cuenca, L.[1], Soto, D.[1,2,3], Comes, N. [1,2,3] and Gasull, X.[1,2,3]

[1] Neurophysiology Lab, Department of Biomedicine, Medical School, Universitat de Barcelona. [2] Institut de Neurociències, Universitat de Barcelona. [3] Institut d'Investigacions Biomèdiques August Pi i Sunyer (IDIBAPS), Barcelona, Spain.

TWIK-Related Spinal cord K+ channel (TRESK), a background K+ channel (K2P family), is expressed in nociceptors and has a main role in setting the resting membrane potential, action potential firing and neuronal excitability. A subset of nociceptive neurons expressing specific transient receptor potential (TRPs) and Mas-related G protein-coupled receptors (Mrgprs) are activated by pruritogens and mediate itch sensations. Because TRESK is key in pain sensitivity, we hypothesize that background K+ channels specifically expressed in pruritogen-activated neurons, are likely to modulate itch sensations and constitute a potential target for the treatment of chronic itch pathologies, including renal or liver failure, Hodgkin's lymphoma and different types of dermatitis.

Bioinformatics analysis indicated that TRESK is expressed in subpopulations of sensory neurons involved in itch generation that contain receptors for chloroquine (MrgprA3), histamine (H1), β-alanine (MrgprD) or BAM8-22 (MrgprC11). We found that different populations of primary cultured sensory neurons from both wild-type or TRESK knockout mice were activated by β-alanine or histamine in calcium imaging experiments. Injection of histamine in the mouse cheek produced a similar itching behavior in both mice groups. In contrast, CQ administration produced a larger effect in animals lacking TRESK compared to wild-type, suggesting that TRESK might be modulating the non-histaminergic itch pathway. In the ocular surface, similar effects for histamine but opposite for CQ were obtained suggesting that this structure presents different mechanisms than the skin. We are currently evaluating the role of TRESK in acute and chronic itch using mice models such as psoriasiform skin inflammation, allergic contact dermatitis and dry skin.

Our data indicate that TRESK is involved in the regulation of the excitability of a subset of sensory neurons mediating histaminergic-independent itch, which have been proposed to have a prominent role in chronic itch diseases, highlighting TRESK as a possible candidate for therapeutic intervention.

29) Differential regulation of AMPARs by auxiliary subunit γ-2 depending on variable stoichiometry

Federico Miguez, Esther Gratacòs-Batlle, David Soto.

Laboratori de Neurofisiologia, Departament de Biomedicina, Facultat de Medicina, Universitat de Barcelona. Institut d'Investigacions Biomèdiques August Pi i Sunyer (IDIBAPS). Institut de Neurociències Barcelona, Spain. Barcelona, Spain.

In the central nervous system, fast excitatory neurotransmission is mainly carried out by AMPA-type glutamate receptors. AMPARs are big complexes formed by several proteins (Schwenk et al, 2012). The ion channel is conformed by 4 AMPAR subunits (GluAs) that confer the receptor the basic properties. However, the AMPAR function is finely tuned by auxiliary subunits. There are several of these modulatory subunits, but TARPs (transmembrane AMPAR regulatory proteins) strongly modulate AMPAR function by regulating the trafficking and gating of neuronal AMPARs (Milstein & Nicoll, 2008). How many auxiliary subunits are present into the GluA structure and whether a distinct number of TARPs modulate AMPARs –stoichiometry – is currently poorly studied. Just a few works have focused on this issue raising heterogeneous conclusions (Shi et al, 2009; Kim et al, 2010; Hastie et al, 2013), most probably reflecting that the AMPAR:TARP stoichiometry varies between neuronal types. It is unknown how many TARPs are needed to provoke significant changes in AMPAR properties.

In this work we have studied how the prototypical TARP γ-2 (stargazin) modulates AMPAR behavior depending on different fixed stoichiometries (2 or 4 TARPed AMPARs). To answer this question, by means of electrophysiology on tsA201 cells we have studied AMPAR gating properties using different fusion proteins of GluAs/γ-2 to obtain AMPARs with fixed amounts of TARPs resembling some specific combinations present in neurons (for example cerebelar granule cells). While most of AMPAR properties are changed differentially depending on the number of TARPs (2 or 4) present into the complex, other properties need a full stoichiometry of 4 TARPs to be altered. It is thought that neurons can have 2 or 4 TARPs per AMPAR complex depending on the brain area, thus the differential stoichiometry will translate in a subtle control AMPAR-mediated integration processes in different neurons of the brain.

30) CPT1C modulates AMPAR trafficking by a depalmitoylating mechanism

Esther Gratacòs-Batlle[1], Nuria Sánchez-Fernández[1], Mireia Olivella[2] & David Soto[1]

[1]Laboratori de Neurofisiologia, Departament de Biomedicina, Facultat de Medicina, Universitat de Barcelona. Institut d'Investigacions Biomèdiques August Pi i Sunyer (IDIBAPS). Institut de Neurociències Barcelona, Spain. [2]Departament de Biologia de Sistemes, Universitat de Vic, Spain.

In neuronal cells, AMPA receptor (AMPAR) gating and trafficking behaviour depends on: the channel pore and the auxiliary subunits. AMPAR trafficking properties are determined by interactions of core-forming transmembrane proteins with a huge amount of other intracellular components. Carnitine palmitoyl transferase 1C (CPT1C) has been described to be in contact with AMPARs in brain extracts. CPT1C is a member of the family of CPT1s, which are involved in fatty acid transport through mitochondrial membranes to allow fatty acid oxidation. However CPT1C is different from other CPT1s. While CPT1A is ubiquitously expressed, CPT1C is almost restricted to neurons where fatty acid oxidation does not represent an important energy source, it shows a different intracellular pattern being located at ER level and is not involved in fatty acid oxidation. We have previously demonstrated that the interaction between GluA1 subunit of AMPARs and CPT1C is important in modulating surface expression of AMPARs. The effect of CPT1C depends in a palmitoylable residue in GluA1 subunit, raising the possibility of a palmitoylation mechanism in this CPT1C-dependent modulation. Here we explore the mechanisms underlying this modulation focusing on possible palmitoylation changes in GluA1 subunit. We have identified by in silico analysis several residues conserved in CPT1C resembling those present in palmitoylthioesterase APT1. Immunofluorescence and electrophysiological experiments with mutations in key residues of CPT1C corroborated the putative role of CPT1C as a depalmitoylating enzyme of GluA1. Inhibiting CPT1C activity with Palmostatin-B confirmed the hypothesis. Finally, the CPT1C effect on GluA1 is isoform specific since CPT1A is not able to increase GluA1-mediated currents.

Posters presentations: Clinical and Applied Psychology

31) Depression and quality of life in the elderly with the SHARE (Survey of Health, Ageing and Retirement in Europe) project

Cristina Portellano-Ortiz; Josep Garre-Olmo; Laia Calvó-Perxas; Josep Lluís Conde-Sala.

Departament de Cognició, Desenvolupament i Psicologia de l'Educació; Secció de Psicologia del Desenvolupament i de l'Educació; Facultat de Psicologia; Universitat de Barcelona

Objectives: The objective of this study is to explore the variables associated with aging that influence the onset and / or maintenance of depressive symptoms and quality of life (QoL) of the elderly.
Method: The study sample (62,800 people between 50 and 104 years of age, in 2013) comes from the SHARE (Survey of Health, Ageing and Retirement in Europe) databases.
Results: Depressive symptoms were more common in participants with poorer perception of quality of life, poor self-perception of physical health, in women and among those who did not participate in social and / or physical activities.
In the global multilevel analysis, greater satisfaction with life, lower depression, economic facilities, better perception of health, physical exercise, absence of functional deficits, lower age and the performance of activities were associated with better QoL in all countries. Higher education was associated with higher QoL only in Eastern and Southern Europe and caring for the grandchildren only in the countries of the South. The Nordic and continental countries had better socioeconomic indicators and better QoL scores, in contrast to the East and South, with worse socio-economic conditions, poorer welfare models, and lower QoL scores.
Conclusions: The worse perception of physical health, the female sex, a worse perception of quality of life and not participating in social and physical activities were predictors of greater depressive symptomatology. Regarding QoL, the scores were in line with the socio-demographic factors of the participants as well as the socio-economic indicators and European social welfare models.

32) ¿What are the implications of Virtual Reality in alcohol use and abuse among college students?

Alexandra Ghita, Joana Pla Sanjuanelo, Ferran Vilalta-Abella, Bruno Porras Garcia, Marta Ferrer-García & José Gutiérrez-Maldonado

University of Barcelona, Department of Clinical Psychology and Psychobiology, Spain

Virtual Reality (VR) has been recently the focus of the current research in clinical and health psychology. Implemented as an assessment or treatment instrument, VR has proven its effectiveness in different psychopathologies such as anxiety disorders, eating disorders, developmental disorders, psychosis or substance use disorders. Numerous studies have demonstrated its long-term positive effects particularly in post-traumatic stress disorder and phobias. Current research aims to give a comprehensive account of the usefulness of VR in assessing and treating craving for alcohol or drugs. In college students, drinking-related behavior represents a public concern with consequences for health and academic performance. The aim of the present study was to determine which measures (behavioral and self-reported measures of craving and anxiety) differentiate best between light- and heavy-drinking college students when exposed to a VR alcohol-cue environment. 25 college students participated in this study, of whom 13 were light drinkers (standard drink units (SDU)/month ≤ 10) and 12 heavy drinkers (SDU)/month ≥ 11). Participants completed the Alcohol Use Disorder Identification Test (AUDIT) before exposure to the VR environment. Heavy drinkers scored higher than light drinkers on AUDIT. The virtual environment consisted of four situations: restaurant, bar, chill-out area, and bedroom, where participants could choose alcoholic or non-alcoholic beverages. An Oculus Rift Development Kit2 headset was used as the Head Mounted Display. In each situation, craving and anxiety were self-reported on a visual analog scale (VAS, from 0 to 10). The results showed differences between groups in the type of beverage chosen in the VR situations, whereby heavy drinkers chose alcoholic drinks more frequently. However, no statistically significant differences were found between groups in craving or anxiety levels reported on the VAS during VR exposure. Heavy-drinking students show a preference for alcoholic beverages in all VR situations compared with light drinkers, but do not experience different levels of craving or anxiety as assessed with VAS. If virtual environments are used to detect heavy drinking cases, behavioral parameters such as choosing between alcoholic or non-alcoholic cues seem more suitable than self-reports of craving or anxiety. This is of great interest particularly in health promotion and prevention of substance use and abuse among college students.

33) Functionality and schizophrenia: the expert's perspective

Laura Nuño

Dep. Psicologia Social i Psicologia Quantitativa, Universitat de Barcelona

Schizophrenia is a chronic mental illness associated with several functional impairments. There has been an increasing interest in the impact of schizophrenia on functioning. This study aims to explore the content validity of the Comprehensive International Classification of Functioning, Disability and Health (ICF) Core Set for schizophrenia, a shortlist of 97 ICF categories that are relevant for describing functioning and disability of people living with schizophrenia, from the expert's perspective.

A broad search of experts in the treatment of people with schizophrenia from around the world was carried out, considering the most involved health professionals in its treatment: psychiatrists, psychologists, nurses, social workers and occupational therapist. For each health profession, it was planned to perform a three-round survey using the Delphi technique. Participants were requested to answer some questions about how functionality is affected in people with schizophrenia and they could participate using one out of the five languages in which the ICF is translated (i.e. English, Spanish, Chinese, Russian, and French). Answers of the first round were linked to categories of the ICF and those categories that reached an agreement more than 75% among the participants in the final Delphi round were selected.

So far, the Delphi study from psychiatrists' perspective has been done. 352 psychiatrists from 65 countries representing all six World Health Organization regions completed the first round. 109 ICF categories reached consensus at the third round. All the Comprehensive ICF Core Set for schizophrenia categories reached consensus except five categories. Therefore, the content validity of the Comprehensive ICF Core Set for schizophrenia from this perspective was largely supported. Further research is needed including other health professionals to obtain new content validity evidences.

34) Gender differences in construction of self and others in psychosis: a study with the repertory grid technique.

García-Mieres, H. [1,2,3]; Feixas G. [1,2]; Furlan, V. [1], Villaplana, A. [3]; López-Carrilero, R. [3]; Grasa, E. [5]; Barajas, A. [4]; Pousa, E. [6] & Ochoa, S. [3]

[1] Universitat de Barcelona. [2] Institut de Neurociències. [3] Institut de Recerca Sant Joan de Déu. [4] Centro de Higiene Mental de Les Corts. [5] Hospital Santa Creu y Sant Pau. [6] Parc Salut de Mar.

Background: The literature in gender differences in psychosis evidences that men have worse negative and less depressive symptoms than women, while women have better social functioning. However, gender issues in self-concept characteristics have received less attention and its relationship with clinical measures is not clear.
Aims: We want to assess gender differences in the perception of self and others, cognitive structure and implicative dilemmas as measured with the Repertory Grid Technique (RGT) in psychotic disorders, and to explore their influence on psychopathological, social functioning, depression and self-esteem variables.
Method: We included 46 adult patients diagnosed with a psychotic disorder from centers of the metropolitan area of Barcelona (Catalonia). The assessment included the RGT, PANSS Lindenmayers' Factors, Social Functioning Scale, Beck Depression Inventory, and Rosenberg's Self-Esteem Scale.
Results: Men showed more negative symptoms and worse social functioning. No differences were found in depression and self-esteem. In RGT measures, men generated fewer constructs to describe self and others, which was influenced by negative and cognitive symptoms, while women perceived themselves more negatively, which was influenced by self-esteem and depression.
Conclusions: Our results support the evidence of previous studies and suggest that gender differences could also be associated to the construction of self and others. The RGT can be a useful measure to appreciate the role of construction of self and others also with people with psychosis. Moreover, our results could help to design gender-specific therapeutic strategies.

35) Propuesta de validación de un conjunto de indicadores para la evaluación de la funcionalidad en la esquizofrenia desde la perspectiva de los pacientes y los cuidadores

Ainoa Mateu, Juana Gómez-Benito, Georgina Guilera y Maite Barrios.

Social Psychology and Quantitative Psychology Department, University of Barcelona.

La funcionalidad es un aspecto básico que se ve afectado en personas con diferentes condiciones de salud, como es el caso de la esquizofrenia. Es necesario un mayor conocimiento de cómo está alterada la funcionalidad en este trastorno y cómo evaluarla y tratarla desde un enfoque multidisciplinar.

Nuestro equipo desarrolló un conjunto de 97 categorías a partir de la "Clasificación Internacional del Funcionamiento, de la Discapacidad y de la Salud" (CIF) elaborada por la Organización Mundial de la Salud (OMS) en 2001 y que constituye el marco teórico universalmente aceptado para la evaluación del funcionamiento. Para que este Conjunto Básico (CB) sea útil es necesario comprobar su validez. La validación del CB sigue un proceso basado en la evidencia que integra información desde distintas perspectivas. Además, se pretende que el CB sea aplicable internacionalmente, por lo que la validación involucra a las seis regiones mundiales de la OMS (África, Mediterráneo oriental, Asia sudoriental, Pacífico occidental, Europa y la región de las Américas).

Para integrar la perspectiva de los pacientes y cuidadores en el proceso de validación se diseñó un protocolo para la realización de grupos focales con pacientes o cuidadores, así como un video demostrativo, que se distribuyó a distintos centros que se ofrecieron a participar en el proceso de validación.

Los grupos focales (GF) son discusiones planificadas para obtener las percepciones sobre un área de interés en un contexto permisivo. Cada GF incluye de 4-7 participantes, un moderador y un asistente. Se realizarán GF en cada región hasta obtener la saturación.

36) Kinship and cohabitation in relation to caregiver burden in the context of Alzheimer's disease: a 24-month longitudinal study

Vanesa Viñas Díez.

Department of Cognition, Development and Educational Psychology of the Faculty of Psychology (University of Barcelona).

Objectives: The aims of the study were to identify the clinical characteristics of three groups of caregivers: spouses, live-in adult–child or non-live-in adult–child, and their relation to the degree of perceived burden (Caregiver Burden Interview).
Methods: The sample comprised 275 Alzheimer's disease primary caregivers, with a follow-up of 24 months. Cognitive, functional and behavioural characteristics were evaluated in persons with dementia, whilst sociodemographic data, use of socio-medical resources, physical and mental health and self-perceived burden were assessed in caregivers. Generalized estimating equations were used for longitudinal data analysis.
Results: Spouse caregivers were 45.0% men, sole caregivers (>80%), used few external resources and had worse physical health. The number of female adult–child caregivers was higher (>75%). The live-in adult–child group, compared with the non-live-in adult–child group, was less likely to be married, had a lower level of education, was more commonly the sole caregiver and used fewer external resources. The greatest burden was observed in live-in adult–child caregivers, and the lowest in the non-live-in adult–child group, with no significant variation in the follow-up for both groups. Spouses had an intermediate level of perceived burden, which rose significantly during follow-up ($p<0.001$).
Conclusions: Kinship and cohabitation with the persons with dementia were associated with different scores and evolution of the burden, with an increase in the follow-up of the spouses, and with more or less burden, depending on cohabitation, in the adult–child groups. Interventions to reduce the level of burden on caregivers should consider these differences.

37) Using Mokken Scaling Analysis for Wechsler Adult Intelligence Scale-Fourth Edition Subscales

Gomaa Said Mohamed Abdelhamid [1, 2], Juana Gómez-Benito. [2, 3]

[1] Fayoum University, Faculty of Education, Department of Educational Psychology, Fayoum, Egypt. [2] University of Barcelona, Faculty of Psychology, Department of Social Psychology and Quantitative Psychology, Barcelona, Spain. [3] Institute of Neurosciences, University of Barcelona, Barcelona, Spain.

Introduction. The Wechsler Adult Intelligence Scale-Fourth Edition (WAIS-IV; Wechsler, 2008) is one of the most well-known tests in the field of adult intelligence assessment. Several recent studies have explored the validity of the WAIS-IV using classical theory, but there are many limitation for the classical theory. Therefore, no study has focused on the WAIS-IV using modern theory, such as Mokken scaling analysis (MSA; Mokken, 1971). The MSA have two models: the monotone homogeneity model (MHM) and the double monotonicity model (DMM).

Aims. This study explores MSA to investigate the psychometric statistics for six subscales of the WAIS-IV consisting of dichotomous items

Materials & Methods. This study uses six subscales of the WAIS-IV in which items are scored 0, 1: visual perception (VP), figure weight (FW), picture complete (PC), matrix reasoning (MR), arithmetic (AR) and information (INF). Two hundred- fifty adults were recruited for this study (156 [62.4%] female and 94 [37.6%] male), aged between 18 and 24 (average 20.65 years, SD 1.71). The R package Mokken V 2.8.2 (Van der Ark, 2016) was used to analyze the data.

Results. The MHM fitted all items of the six subscales, although a small number of items showed poor fit as measured by the scalability coefficient. Surprisingly, DMM was a good fit for all items of the VP, MR, FW and AR subscales, while backward item selection method only deleted two items from the PC and INF subscales that violated the non-intersection assumption.

Conclusion. In conclusion, analysis of WAIS-IV using a variety of MSA models provides novel, accurate and relevant information.

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