SAN

SOCIEDAD ARGENTINA DE
INVESTIGACIÓN EN NEUROCIENCIAS

Argentine Society for Research in Neurosciences

Abstracts of the 2019 Meeting of Argentine Society for Research in Neurosciences

XXXIV ANUAL MEETING SAN 2019

VILLA CARLOS PAZ

CÓRDOBA

ARGENTINA

OCTOBER 3-5, 2019

The 2019 meeting of the Argentine Society for research in Neurosciences (SAN) was held at Villa Carlos Paz, Córdoba, Argentina, in Portal del Lago Hotel, from October 3rd to 5th 2019.

There were 350 attendees among researchers, scholars, PhD students and guests from different centers and universities of Argentina and abroad from 8 countries of Latin America, North America and Europe. Our congress had a total of 4 (four) Plenary Lectures, 6 (six) Symposia, 2 (two) Short Conferences, 6 (six) Youth Conferences, 19 (nineteen) Oral Communications, 256 Posters coveringa broad number of areas in the field of neurosciences together with 2 (two) special activities at lunch time and a round table on "Gender and Science".

It is noteworthy that two of the Plenary Lectures were placed in honors of the pioneers of neurochemistry andneurobiology of Argentina, Drs. Ranwel Caputto andEduardo De Robertis. This year the “Ranwel Caputto” Lecture was delivered by Prof. Belen Elgoyhen of the University of Buenos Aires (Argentina) and the “De Robertis” Lecture by Prof. Beatriz L. Caputto of the National University of Córdoba (Argentina). The “Opening Lecture” was given by Prof. Marla B. Feller, Department of Molecular and Cell Biology and Helen Wills Neuroscience Institute, University of California (USA) and the “Hector Maldonado” Lecture by Prof. Lucas Pozzo-Miller Department of Neurobiology, University of Alabama at Birmingham (USA). Short conferences were delivered by Drs. Ethan Buhr of the University of Washington in Seattle (USA), and Emilio Kropff of the Leloir Institute, Buenos Aires (Argentina).

As pre-meeting activity, the specific course for PhD students “Molecular and Cellular Neuroscience and Neurochemistry: Experimental strategies for studying the nervous system in health and disease”, took place on September 30-October 1-2, 2019 at the School of Chemical Sciences of the National University of Córdoba, Córdoba with the participation of more than 60 students.

Remarkably, all the activities organized, including the Symposia and the Young Investigator Lectures, covered a number of diverse disciplines in the field of neurosciences with the participation of outstanding invited speakers from Argentina and other countries.

Moreover, a very friendly atmosphere for discussion and data presentation was generated during the poster and oral communication sessions with the participation of 104 researchers, 139 Ph.D. students, 64 undergrads and 34 postdocs from Argentina**,** Chile**,** Brazil, Uruguay, USA, Canada, Denmark, Germany and France.

Short Program SAN 2019

Mon., Sept 30th - Wed., Oct 2nd	Thursday, October 3rd		Friday, October 4th	Saturday, October 5th
9:00 - 18:00 PRE-CONGRESS COURSE *"Molecular and Cellular Neuroscience and Neurochemistry: Experimental strategies for studying the nervous system in health and disease"* *Auditorio Ciencias I Facultad de Ciencias Químicas – UNC* **REGISTRATION**	**8:30 - REGISTRATION** **9:00 - 11:00** **SYMPOSIUM I** ***"New perspectives and mechanisms underlying neurological disorders"***		**8:30 - 10:30** **SYMPOSIUM III** ***"Molecular mechanisms of epigenetics and chromatin remodeling during brain development and aging"***	**8:30:00 - 10:30** **SYMPOSIUM VI** ***"Sensory processing and integration in olfactory and tactile systems"***
	11:00 - 11:30 Coffee break		**10:30 - 11:00** Coffee break	**10:30 – 11:00** Coffee break
	11:30 - 12:30 **OPENING LECTURE** Prof. Marla Feller		**11:00 - 13:00** **SYMPOSIUM IV** ***"First impressions: New roles for perinatal factors governing brain development"***	**11:00 - 12:00** **Oral Communications** Room Auditorio (OC 8-12) Room Lago (OC 13-18)
				12:00 - 13:00 **EDUARDO DE ROBERTIS LECTURE** Prof. Beatriz Caputto
	12:30 – Lunch with activities *"The 3Rs in neuroscience research"*		**13:00** - Lunch with activities *"HD Foundation"*	**13:00** – Farewell Lunch
	14:30 - 15:30 **SHORT LECTURES** Ethan Buhr Emilio Kropff	**14:30 – 16:00** **Oral Communications** Room Lago (OC 1-7)	**14:30-15:30** **Young Investigator Lectures** Room Auditorio (YIL 1-3) Room Lago (YIL 4-6)	
	15:30-16:00 **Gender and Science** Verónica de la Fuente			
	16:00 - 17:30 **SYMPOSIUM II** ***"Advances in early diagnosis and in experimental therapy of Alzheimer´s disease"***		**15:30 - 17:30** **SYMPOSIUM V** ***"Sexual differences on development and function of CNS"***	
	17:30 - Coffee break		**17:30** - Coffee break	
	17:30 - 19:30 **Poster Session (Even numbers)**		**17:30 - 19:30** **Poster Session (Odd numbers)**	
	19:30 - 20:30 **RANWEL CAPUTTO LECTURE** Prof. Ana Belén Elgoyhen		**19:30- 20:30** **HÉCTOR MALDONADO PLENARY LECTURE** Prof. Lucas Pozzo-Miller	
	20:30 **WELCOME RECEPTION**		**20:30** **SAN General Assembly**	

Instituto de Investigación Médica Mercedes y Martin Ferreyra, (INIMEC-CONICET-Universidad Nacional de Córdoba), Córdoba, Argentina
Presenting author: ***Josefina Inés Martín,*** *joinesmartin@gmail.com*

Neurons are highly polarized cells typically extending a long thin axon and multiple short branched dendrites. These specialized compartments are developed through the coordination of cellular and molecular mechanisms in order to ensure the proper functioning of the nervous system, and are highly regulated by several small Rho GTPases with their effectors controlling different aspects of neuronal morphology. Among others, these events include actin and microtubules cytoskeleton assembly, and the addition of membrane in neuron specialized regions. Even though most of studies have been focused on classical Rho GTPases (RhoA, Rac1 and Cdc42), other less studied members of this family such as RhoD suggest to have unique effects on cytoskeleton and membrane dynamics. In this study we have analyzed the role of RhoD during the development of axonal polarization and neurite extension. We have also designed and characterized an unimolecular activity RhoD biosensor based on resonance energy transfer (FRET) in order to study the space-time dynamics of this Rho GTPase in fibroblast and neuron cells. Finally, we have evaluated how RhoD affects different dynamic parameters of microtubules cytoskeleton.

Cellular and Molecular Neurobiology

P68.-Passive immunization with anti-Myelin-associated glycoprotein antibodies induces an autistic phenotype associated with altered postnatal neurodevelopment

Mara Soledad Mattalloni[1], Anabela Palandri[1], Cristian Bacaglio[1], Victor Amaya[1], Ronald L Schnnar[2], Pablo H H Lopez[1]
[1] Instituto Martin y Mercedes Ferreyra INIMEC CONICET UNC,
[2] Department of Pharmacology and Molecular Sciences, The Johns Hopkins School of Medicine, Baltimore, USA
Presenting author: ***Mara Soledad Mattalloni,*** *marsol214@hotmail.com*

High titer of anti-Myelin-associated glycoprotein antibodies (anti-MAG mAb) has been observed in infants with autism spectrum disorders (ASDs). We analyzed the possible contribution of MAG to the pathogenesis of ASD by passive immunization with a function-blocking anti-MAG mAb in wild type mice at perinatal stages. Anti-MAG mAb treatment was associated with development of an autistic phenotype characterized by alterations of social interaction and social recognition behaviors, reduced ultrasonic vocalizations and increased stereotyped behaviors with preservation of motor skills. Morphometric stereological analyses showed altered cerebellar neurodevelopment characterized by a transient increase in the number of granule cells at P7 in lobes VI/VII (but not at lobe X) followed by increased neurodegeneration at P14-21. Similarly, we observed a preferential increase of Purkinje cells number in lobes VI/VII characterized by a dystrophic morphology, although their number remained constant along the study. Morphometric studies also identified alterations in cell/size number in prefrontal cortex. MAG-null mice displayed behavioural and neurodevelopmental alterations closely resembling anti-MAG-treated mice. Our results show that blocking MAG function induces phenotypic behaviors and neurodevelopmental alterations associated with ASDs. Altogether, our data supports a pathogenic role of anti-MAG autoantibodies as a leading cause for the development of ASDs in a clinical subgroup.

Cellular and Molecular Neurobiology

P69.-Functional and structural characterization of human heteromeric 5-HT3 receptors

Albano Mazzarini Dimarco[1,2], Cecilia Bouzat[1,2], Jeremías Corradi[1, 2]

[1] *Instituto de Investigaciones Bioquímicas de Bahía Blanca – INIBIBB*
[2] *Departamento de Biología, Bioquímica y Farmacia, Universidad Nacional del Sur*
Presenting author: ***Albano Mazzarini Dimarco,*** *amazzarini@inibibb-conicet.gob.ar*

5 HT3 receptors are the only serotonin (5-HT) receptors that belong to the Cys-loop receptor family. They mediate fast excitatory transmission in central and peripheral nervous system. Five different subunits (A-E) have been identified in humans. The A subunit is able to form homomeric receptors (5-HT3A), and it can combine with the B subunit to form heteromeric receptors. We here evaluated if the C-E subunits can combine with the A to form heteromeric receptors. To this end, we constructed a high-conductance A subunit (AHC) that allowed to obtain single-channel events. After expression of the AHC we observed events with an amplitude of ~4.7 pA corresponding to the 5 HT3AHC receptor. However, when AHC was expressed in combination with one of the C-E subunits, events with different amplitudes were detected, thus confirming the expression of heteromeric receptors. From macroscopic currents we observed an increase in the 5-HT EC50 value for each of the heteromeric receptors with respect to that for the 5 HT3AHC receptor. In-silico studies provided insights into the contribution of the different subunits to the 5-HT binding site. Our results demonstrate that C-E subunits can combine with the A subunit to form heteromeric receptors. These results bring structural and functional details about the different human 5-HT3 receptors and will contribute to the development of selective therapies targeting this receptor family.

Cellular and Molecular Neurobiology

P70.-In vivo effect of a single dose of mesenchymal stem cells exogenously expressing IGF-I in a spinal cord compression model

Graciela L Mazzone[1], Samuel Cockey[2], Rocio Bertossi[1], Esteban J Fiore[3], Romina Sierra[1], Guillermo D Mazzolini[3], Jorge B Aquino[1]
[1] *Developmental Biology & Regenerative Medicine Laboratory, Instituto de Investigaciones en Medicina Traslacional (IIMT), CONICET-Universidad Austral, Derqui-Pilar, Buenos Aires, Argentina*
[2] *College of Liberal Arts and Sciences, University of Florida, Gainesville, FL, USA*
[3]*Gene Therapy Laboratory, IIMT, CONICET, Universidad Austral*
Presenting author: ***Graciela Lujan Mazzone,*** *gmazzone@austral.edu.ar*

An important aim in nervous system repair is to develop strategies that avoid main consequences of spinal cord injury by restricting tissue damage, neural death and locomotor function loss. In this regard, application of mesenchymal stem cells as vehicles of gene therapy could be a useful approach. In this study, a compression injury model was developed by applying an aneurysm clip to the spinal cord of BALB/c mice at the thoraco-lumbar level for 1 min. Mesenchymal stem cells, transduced with an adenovirus containing rat IGF-I (AdIGF-I-MSCs) or GFP (AdGFP-MSCs) gene sequences, were injected at the core of the affected zone 7 days post-injury. Locomotor behavioural and histological studies were performed. Locomotion was evaluated in an open field by applying the Basso Mouse locomotor scale rating and foot printing analysis. No apparent changes among experimental conditions were observed with regards glia cells in broad areas within the injury zone. Our preliminary data suggest an improvement in locomotion task and coordination walking pattern after AdIGF-I-MSCs application. This outcome might involve a modulation and/or plasticity of spinal circuits and locomotor neuronal networks.
Supported by Austral University and CONICET.

Cellular and Molecular Neurobiology