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**	

amygdala (BLA) and the lateral entorhinal cortex (LEC). To understand the contribution of the BLA and LEC to the processing of odors we study their functional connectivity to the posterior PC (pPC). We infected the BLA and the LEC with adeno-associated virus to express channelrhodopsin (ChR2-AAV) in either excitatory neurons (under CamKIIa promoter) or inhibitory Parvalbumin interneurons (using PV-Cre mice). We recorded then, in acute brain slices, postsynaptic currents and spiking in different principal neurons of the pPC in response to photostimulation. We found that both excitatory and inhibitory long range projections coming from the BLA synapse preferentially onto pyramidal neurons of the deep layers of pPC and do not contact semilunar neurons of the superficial layer. Moreover, we discover that inputs from both BLA and LEC can modulate the output of pPC neurons in response to stimulation of OB afferents. The LEC and BLA inputs could provide contextual and valence information associated to odors. To investigate the role of those regions in the processing of odors in vivo, we are conducting experiments to photoinactivate them alternatively during an associative odor-context-reward task and evaluate the effect of that manipulation on the behavior.

Neural Circuit Physiology

P212.-Cortical spiking activity entrainment with beta oscillations is enhanced after nigrostriatal degeneration and when L-DOPA effects have worn off

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Abnormal involuntary movements known as L-DOPA-induced dyskinesia (LID) are a common complication in Parkinson's disease (PD) after prolonged treatment with L-DOPA, which is the gold standard medication. Little is known about the oscillatory activity associated with LID, especially in the motor cortex (MC). However recent studies show that exaggerated beta activity (15–35 Hz) who emerge in the basal ganglia after nigrostriatal degeneration, correlate with motor impairment in PD and can be suppressed by LID. Our previous characterization in MC disclosed a similar pattern, with an increased number, duration and power of beta events. Interestingly this pattern was reverted during the acute effect of L-DOPA, but reappeared when L-DOPA effects have worn off. Here we sought to identify cortical neuronal populations related to this rhythm. We performed recordings of single unit activity by means of high density electrodes in primary MC of parkinsonian mice before and after L-DOPA regime that induced LID. We found an increased mean firing rate in both conditions. Also, phase preference of spiking activity to beta oscillations was higher in lesioned than in sham animals. This pattern was present both in putative pyramidal neurons and interneurons. These results reveal a better entrainment of neuronal activity with beta oscillations in the parkinsonian condition, which is not reversed by chronic L-DOPA administration, and could explain the increased beta power previously observed.

Neural Circuit Physiology

P213.-Physiological significance of the KCNQ4-mediated M-current in the pedunculopontine nucleus of the reticular activating system

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The pedunculopontine nucleus (PPN) is part of the reticular activating system (RAS) which is associated with sleep regulation. The PPN has cholinergic and non-cholinergic neurons. A hallmark of the PPN-cholinergic neurons is the M-current, a slowly activating, non-inactivating voltage-gated potassium current. KCNQ2 to 5 subunit alone or in combination are responsible for the M-current. Our aim was to investigate the contribution of the KCNQ4 subunit to PPN neuronal function. We used a transgenic mouse model for KCNQ4 (knock-out (KO)) and one with fluorescent-labeled cholinergic neurons (tdTomatoStop+ChAT::Cre). We analyzed KCNQ4 expression by real-time PCR and its localization using immunofluorescence. We also studied the M-current by electrophysiology on brain slices, the contribution of KCNQ4 to neuronal activity and its influence on circadian rhythm. We found a weak mRNA expression of KCNQ4 in PPN and the protein was located only on cholinergic neurons of the external limits of the nucleus. M-current was present in most of cholinergic neurons in WT animals, but absent in 40% of them in the KO ones. These last also exhibited behavioral alterations in the activity cycles showing a 5-hour increase and a higher sensitivity to changes in the light/darkness cycles. In summary, we found that only a subpopulation of PPN cholinergic neurons have KCNQ4-dependent M-current and this subunit contributes to modulate the circadian rhythm through the activity of the RAS system.

Neurochemistry and Neuropharmacology

P214.-Protective effects of imidazolium salts in C. elegans models of stress and neurodegeneration

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In this work, using an established model in biomedical research, the nematode C. elegans, we synthesized imidazolium salts and performed a screening to analyze their ability to improve oxidative stress (OS) resistance. We identified a derivate, 1-Mesithyl-3-(3-sulfonatopropyl)imidazolium (MSI), that enhances animal resistance to OS. To delineate MSI roles, we split this work into two goals: i) to describe MSI action mechanisms and, ii) to evaluate MSI role in neurodegenerative models. To gain insight into its mechanism of action, we evaluated MSI ability to activate DAF-16 (FOXO in vertebrates), a transcription factor relevant for cytoprotective defense mechanisms. Unexpectedly, our experiments revealed that MSI stress protection was not dependent on DAF-16. These results support the idea that other transcription factors (such as SKN-1 (Nrf-2 in vertebrates), HSF-1), could be involved in MSI protection. The second goal is held by the theory that links OS to aging and neurodegeneration. We are currently evaluating if MSI increases lifespan, healthspan, and improves biological markers of neurodegeneration in a C. elegans model of Alzheimer disease. This strain expresses Aβ1-42 in muscle and shows age-dependent protein aggregation and paralysis. Our preliminary results show that MSI delays paralysis in this strain. Additional research is needed to underpin the protective role of MSI and to determine if these effects can be extrapolated in other neurodegenerative scenarios.

Neurochemistry and Neuropharmacology

P215.-Neuroprotective effect of melatonin loaded in ethylcellulose nanoparticles applied topically in a retinal degeneration model in rabbits