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**	

ABSTRACTS INDEX

contrast, 10 µM RA inhibited PDGFRα+ cell proliferation. However, combined treatment of 5 µM PIO + 10 µM RA exerted effects comparable to those of single PIO treatment. In turn, in OPC cultures, 10 µM RA treatment revealed a differentiating effect on PDGFRα+ cells and an increase in their morphological complexity. These results suggest the participation of RXRγ and PPARγ in OPC proliferation and differentiation and may be thus considered possible therapeutic targets in the treatment of demyelinating diseases.

Cellular and Molecular Neurobiology

P83.-Intracellular trafficking of Diacylglicerol Lipase in Kinesin Light Chain knock-out neurons

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Endocannabinoid (eCB) signaling modulates axonal growth, guidance and synaptogenesis. Diacylglycerol lipase-α (DAGL-α) and -beta (DAGL-β) synthesize 2-arachidonoyl-glycerol (2-AG). 2-AG regulates the axonal growth cone-turning decision through the activation of the cannabinoid type 1 receptor (CB1R). Acquisition of precise neuronal connectivity requires a proper targeting and accurate spatiotemporal localization of both, CB1R and DAGL, in the surface of navigating axons.Although cargo delivery mediated by molecular motors is essential in developing neurons, the transport mechanism of the eCB system in developing axons is not fully understood. Our previous results showed significant impairment in CB1R axonal transport properties in neurons lacking the kinesin light chain 1 (KLC1) subunit of the anterograde motor kinesin-1. Defects in CB1R axonal transport triggers dysfunctions in eCB-dependent axonal growth. Here, we tested whether KLC1 deletion also affects the intracellular trafficking of DAGL-β. By live-cell imaging of fluorescent DAGL-β tagged vesicles we characterized the axonal transport properties of DAGL in transfected primary hippocampal neurons. Our preliminary data reveals that DAGL-β vesicles moves at slower speeds than CB1R vesicles and that KLC1 deletion impairs anterograde DAGL-β average velocity without affecting retrograde velocity. These results suggest that kinesin-1 could play a key role in both, DALG and CB1R intracellular trafficking.

Cellular and Molecular Neurobiology

P84.-Neuroprotective effect of FK506 against oxidative stress

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Immunophilins FKBP51 and FKBP52 bind the macrolide FK506. Previously, we reported that FK506 favours neurodifferentiation and the neuroregeneration. Here, we analyzed whether FK506 also shows neuroprotective action to oxidative stress. A rapid neuritogenesis was observed in undifferentiated N2a cells treated with 1 µM FK506 in the absence of trophic factors, including serum. Then, 250 µm slices from prefrontal cortexes from Balb-C mice (60 d) were prestabilized for 72 h and incubated for 4 h with 200 µM H_2O_2. Western blots revealed the induction of Hsp90, Hsp70, FKBP52 and p23, which was prevent by 1 h pretreatment with 1 µM FK506. While controls showed three phosphorylated isoforms of FKBP51, treatment with H_2O_2 only exhibited the least phosphorylated band. In turn, pretreatment with FK506 protected the phosphorylated isoforms, and treatments

with FK506 alone showed the intermediate phosphorylated band (reactive to anti-P-Tyr IgG), suggesting that this isoform may be responsible for the mechanism of action of the drug. Hypoxia was generated by stereotactic injection of 2 µl 50 mM CoCl2 in the prefrontal cortex of the right hemisphere, and the contralateral was used as control. The overexpression of chaperones was partially impaired by pretreatment with FK506. Rotarod and open field (AnyMaze) studies evidenced better and faster recovery of FK506-treated mice. This study shows for the first time the neuroprotective effect of FK506 against oxidative stress in nervous tissue.

Cellular and Molecular Neurobiology

P85.-SARA involvement in modulating TGFβ signaling during neural development

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Several events are necessary for proper neuronal development, such as cytoskeleton dynamics and endosome trafficking. SARA is a protein that binds to early endosomes; performing both traffic and signaling functions, as in the transforming growth factor β (TGFβ) pathway. In this sense, it has been described that SARA recruits Smad2/3, favoring the activation of this pathway; but it can also modulate the inactivation of TβRI by PP1c in both epithelial cells and cell lines. In addition, TGFβ signaling has been shown to specify the axon during neuronal development; however, the participation of SARA in this signaling pathway during development remains unknown. For this reason, we proposed to analyze the role of SARA in TGFβ signaling during neuronal development. Results obtained in cultures of hippocampal neurons, by FRET showed physical interaction between SARA and TβRI. In addition, performing experiments of loss and gain of function, we found that dominant-negative form of SARA (SARA-F728A) generates greater axonal growth and loss of axonal specification compared to control condition. Interestingly, this mutant alters its binding to the PP1c protein, keeping the TGFβ pathway over-activated. Also by FRET, we find that SARA-F728A has more interaction with PP1c and GADD34 than control, suggesting that SARA prevents TβRI dephosphorylation. These results suggest that SARA negatively modulates the TGFβ pathway, which seems to be a necessary requirement for proper axon specification.

Cellular and Molecular Neurobiology

P86.-Spontaneous electrical activity regulates axonal arbor growth in developing Zebrafish lateral line afferent neurons

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Neuronal circuits responsible for processing sensory information are established early in development through a combination of genetic programs and activity-dependent processes. A remarkable feature of this process is that it relies on stimulus-independent or “spontaneous” electrical activity (SEA) generated within sensory organs. In order to decipher the mechanisms by which SEA affects the assembly of developing sensory circuits, we used the Zebrafish (Danio rerio) lateral line system (LL). The LL allows fishes and amphibians to detect water motion