

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 covering a 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 and neurobiology of Argentina, Drs. Ranwel Caputto and Eduardo 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.

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Short Program SAN 2019

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

P252.-Intracellular modulation of α7 ionotropic and metabotropic functions by tyrosine phosphorylation Juan Facundo Chrestia¹, Ariana Bruzzone², María del Carmen Esandi¹, Cecilia Beatriz Bouzat¹

The $\alpha 7$ receptor is a nicotinic receptor present in neuronal and non-neuronal cells. $\alpha 7$ acts as a ligand-gated ion channel and as a metabotropic receptor. We investigated the role of tyrosine phosphorylation of the intracellular domain (ICD) in the dual ionotropic/metabotropic receptor function. Single-channel recordings from HEK cells expressing $\alpha 7$ showed that channel activity appears as brief isolated openings and episodes of few openings in quick succession (bursts). Exposure to an inhibitor of Src family kinases (PP2) increased the frequency and duration of bursts while preincubation with an inhibitor of tyrosine phosphatases had the opposite effect. Co-expression of $\alpha 7$ and an inactive Src kinase also increased burst duration. A mutant $\alpha 7$ lacking tyrosine phosphorylation sites in the ICD showed longer burst durations and insensitivity to PP2, thus recapitulating the effects of phosphorylation inhibition on wild-type $\alpha 7$. Cells exposed to the specific $\alpha 7$ agonist (PNU-282987) showed an increase of ERK1/2 phosphorylation, which was abolished by exposure to a tyrosine kinase inhibitor. PNU-282987 did not trigger ERK phosphorylation in cells expressing the mutant receptor lacking tyrosine residues or co-expressing $\alpha 7$ and $\alpha 7$ -ICD domain. Our results indicate that dephosphorylation positively modulates ionotropic $\alpha 7$ activity in a way compatible with decreased desensitization, and that the phosphorylated state of $\alpha 7$ -ICD plays a role in metabotropic receptor responses.

Synaptic Transmission and Excitability

P253.-Histamine-enhanced ASIC mediated currents contribute to anterior cingulate cortex long-term potentiation

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Acid-sensing ion channels (ASICs) are H+-gated channels belonging to the ENaC/Deg superfamily that are involved in synaptic transmission and in neurodegenerative diseases. During synaptic transmission, acidification of the synaptic cleft due to the co-release of neurotransmitter and H+ from synaptic vesicles activates ASIC channels in mice. We used slices from the anterior cingulate cortex (ACC) of P30-60 postnatal mice to evoke glutamatergic AMPA receptor-mediated excitatory postsynaptic currents (EPSCs), recorded in whole-cell patch-clamp at layer I pyramidal neurons. After blocking AMPA, NMDA, GABA and glycine receptors, we detected ASIC mediated synaptic currents (ASIC-SCs) sensitive to ASIC-1a inhibitor psalmotoxin-1.ASIC-SCs were enhanced by the neuromodulator histamine, which specifically modulates homomeric ASIC-1a channels, as well as by corticosterone. Long-term potentiation (LTP) is a major type of long-lasting synaptic plasticity and is associated with learning, memory, development and neuropathic pain. Neurons in the ACC play critical roles in chronic pain. LTP was induced by theta burst stimulation (TBS) of the callosal afferents. Extracellular field EPSP and whole-cell patch-clamped EPSC recordings demonstrated that ASIC-SCs contribute to ACC LTP induction. Stimulated by a TBS below threshold, glutamatergic synapses undergo LTP by the potentiating effect of histamine on ASIC channels, which is prevented by previous incubation with psalmotoxin-1.

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