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**	

(WT) mice. We found that CR WT mice lost 25% of their body weight after 5-day of CR, and that displayed a 5-day compensatory hyperphagia after refeeding. As compared to ad libitum fed mice, CR mice showed 1) higher plasma ghrelin levels, 2) higher density of AgRP+ fibers in several brain areas and 3) a matched increase of the levels of the marker of neuronal activation c-Fos in those brain areas. To test if these effects require the presence of the ghrelin receptor (GHSR), we studied CR GHSR-deficient mice. We found that CR GHSR-deficient mice lost ~25% of BW after 5-day 60% CR, similar as seen in WT mice, but showed a significantly smaller compensatory hyperphagia after refeeding. Thus, we conclude that ghrelin/GHSR system is involved in the regulation of the compensatory hyperphagia in CR mice.

Neuroendocrinology and Neuroimmunology

P233.-Dissecting the immune response in the CNS: astrocytic response to interaction with leukocytes

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The old paradigm on CNS immune privilege has been reformulated and a role for peripheral immune system in CNS immune processes is accepted, although not completely understood. The view of astrocytes physiology has also shifted. There is a general consensus in that astrocyte population shows high degree of heterogeneity, as does its response to different pathological contexts. A more detailed comprehension of the response of CNS local immune cells to its peripheral counterparts is crucial for a global understanding of neuropathological processes. In the present work we used an in vitro co-culture model to study astrocytic response. To achieve this, we cocultured rat primary glial cells with acutely obtained leukocytes from naïve or rats exposed to brain ischemia which models stroke. Our results showed that when enriched astrocytic culture (95-98% GFAP+) contacted leukocytes (from either naïve or ischemic rats) there were no significant alterations in GFAP immunoreactivity reactivity or morphological phenotype. However, astrocytic cellular retraction and reorganization was evident, and scar-like structures were seen when leukocytes contacted mixed glial primary cultures that include microglia. Moreover, fixed leukocyte also induced these scar-like structures, indicating that surface molecules present in leukocytes might be enough to cause them. Using leukocytes from ischemic rats did not show significant differences.

Neuroendocrinology and Neuroimmunology

P234.-Neural modulation of systemic stress response requires the insulin like-peptide INS-3 in C. elegans

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Perpetuation of the flight response inhibits defensive cytoprotective mechanisms, leading to early onset of age-related disorders from invertebrates to mammals.We have recently shown that, in C. elegans, the flight response induces neuronal release of Tyramine (TA, invertebrate analog of adrenaline), that stimulates the adrenergic-like receptor TYRA-3 in the intestine. This leads to the activation of the DAF-2/Insulin/IGF-1 pathway in non-intestinal cells and the inhibition of cytoprotective mechanisms. However, the signals that link the activation of TYRA-3 in the intestine with the DAF-2 insulin receptor in other tissues is unknown. We, therefore,

performed a screening of Insulin like-peptides expressed in the intestine by RNAi and identified that lack of ins-3 improves resistance to oxidative and thermal stress. This resistant phenotype cannot be reversed by exogenous TA and it is mediated, at least partially, by DAF-16/FOXO. Moreover, by using genetics we found that tyra-3 and ins-3 act in the same pathway. In addition, we found that only the intestinal rescue of ins-3 null mutants was able to restore the resistance to wild-type levels. We propose that INS-3 could be the signal molecule that connects the intestine, where TA receptor is expressed, with distal tissues. Given the high degree of conservation of fundamental mechanisms, this work can contribute to the understanding of neurohormonal coordination of stress responses in animals.

Neuroendocrinology and Neuroimmunology

P235.-Increased pro-inflammatory response in a mouse model of neurodevelopmental disorder

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Rett Syndrome (RTT) is a pervasive developmental disorder caused by mutations in methyl-CpG binding protein 2 (MeCP2), a ubiquitous transcriptional regulator. The goal of our project is to evaluate the role of MeCP2 in immune responses in vivo, using an animal model of Multiple Sclerosis (EAE) as an autoimmune challenge and in vitro, using bone marrow derived macrophages (BMDM). Male MeCP2 WT and MT mice, were immunized with MOG 35-55 peptide, scored daily for EAE symptoms and sacrificed at 12 dpi (acute stage) or at 30 dpi (chronic stage). We found that MT-EAE mice showed an accelerated onset of the disease and more severe clinical scores, accompanied by increased infiltration of lymphocytes in spinal cord. Also, we detected a sustained higher expression of TNFα and IFNγ in spinal cords from MT-EAE animals during chronic stage compared to WT. Next, we assessed the response of BMDM from WT and MT mice stimulated with either pro- or anti- inflammatory stimuli. M1-polarized BMDM-MT showed increased expression of TNFα, while M2-polarized BMDM-MT, presented lower levels of FIZZ1, IL10, and CD206 compared to BMDM-WT. Also, independently of the stimuli, BMDM-MT showed higher levels of superoxide production. Thus, MeCP2 mutation appears to bias the response toward a pro-inflammatory profile.Overall, our results suggest that MeCP2 has an active role in maintaining the immune homeostasis in vivo and regulating the immune response in vitro.

Sensory Systems

P236.-Non image forming visual system alterations induced by experimental opticneuritis: therapeutic effect of melatonin

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Optic neuritis (ON) is an inflammatory condition of the optic nerve (OpN), which leads to retinal ganglion cell (RGC) loss. A subset of RGCs expressing the photopigment melanopsin regulates non-image-forming (NIF) visual system functions such as pupillary light reflex (PLR) and circadian rhythms. ON can be induced by a single microinjection of bacterial lipopolysaccharide (LPS) into the OpN. We analyzed the effect of ON on the NIF visual