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**	

cerebellum, induced by the stereotaxic injection of lipopolysaccharides (LPS), results in a decrease in sociability 24 hours after. This effect is prevented completely by dexametasone and partially by ibuprofen. The aim of this project is to identify mediators of these effects by detecting inflammatory molecules that can alter sociability in ASD. To perform this, groups of adult male CF1 mice were injected with either 10ng LPS (LPS group) or saline (SAL group) in cerebellum lobule 7. 30 minutes before, both groups will receive dexamethasone, ibuprofen, or saline. Thus, the experimental design evaluates the effects of drugs (DEXA; IBU; SAL) on the treatment (LPS; SAL). In these animals, we observed that DEXA has a more pronounced effect than IBU in blocking microglia activation. We will further characterize the response, by identifying the expression of pro and anti-inflammatory cytokines in the cerebellum of LPS injected and anti-inflammatory treated mice. To this aim, we will quantify the expression of pro and anti-inflammatory cytokines by RT-PCR. The goal of this work is the identification of possible therapeutic targets for individuals with ASD.

Cellular and Molecular Neurobiology

P100.-Shiga toxin 2 (Stx2) from enterohemorrhagic Escherichia coli (EHEC) produces cerebellar impairment of the vascular unit with inflammatory involvement

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Stx2 from EHEC produces Hemolytic Uremic Syndrome and neurologic alterations including cerebellar involvement in patients. The aim of this study was to determine the mechanisms by which Stx2 causes cell damage in the cerebellum. Mice were injected intravenously with 1ng of Stx2 or 100 µl of saline. Fixed cerebellums were subjected to staining with lectins (microvasculature profile) and immunofluorescence with anti-GFAP (astrocytosis marker) and anti-MBP (myelin protein marker). ELISA kits measured TNFα and IL-10. Stx2 reached the Purkinje and granular layers. Stx2 significantly: decreased the area occupied by the microvasculature (12.58 ± 0.73 Control vs 7.71 ± 0.72 Stx2, day 2, in µm2); increased the expression of GFAP (11.3 ± 0.3 Control vs 17.97 ± 0.87 Stx2, day 2, and 12.11 ± 0.67 Control vs 14.8 ± 0.6 Stx2 day 4, in IOD),and decreased the expression of MBP (67.6 ± 2.4 Control vs 35.4 ± 0.9 Stx2, day 2, and 62.6 ± 2.5 Control vs 46.2 ± 1.4 Stx2 day 4, in IOD); $p<0.001$. Stx2 increased the expression of TNFα at day 2 (4.8 ± 1.7 Control vs 12.4 ± 2.1 Stx2, in pg/mg protein), while IL-10 expression was increased at day 4 (25.05 ± 3.9 Control vs 67.2 ± 10.8 Stx2, in pg/mg protein); $p<0.001$. Finally, Stx2 damaged the cells that integrate the vascular unit, with inflammatory involvement. Further studies are being conducted to elucidate the observed cell events.

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Cellular and Molecular Neurobiology

P101.-Expression and cellular function of KCNQ channels in the eye

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KCNQ subunits (1 to 5) form voltage-gated potassium channels. They are responsible of the M-current that regulates neuronal excitability in the CNS. KCNQ4 and -5 expression has been reported in the retina where could participate in the visual processing. Our aim was to investigate the expression and function of KCNQ channels in different tissues of the eye, employing KO mice for subunits 3 to 5. By KO-controlled immunofluorescence, we found a weak labeling of KCNQ4 in retinal pigmented epithelium (RPE) cells and the ciliary body (CB). KCNQ4 signal in RPE was stronger in the area adjacent to the CB and located in the apical membrane of the cells. In the CB the signal was placed in the basal membrane of the pigmented epithelium (PE) cells. KCNQ5 was found neither in the retina nor RPE or CB. Finally, KCNQ3 was observed in non-pigmented epithelium (NPE) cells of the CB. Gene expression analysis by RT-PCR showed that all KCNQ subunits were present in RPE/retinal tissue while in CB were only KCNQ1, -4 and -5. This profile was altered in KCNQ4 KO mouse with the appearance of KCNQ2. By patch-clamp, we observed the M-current in 40% of the RPE cells while it was absent in the KCNQ4 KO mouse. In CB, the M-current was present in NPE cells, however it could not be analyzed in PE cells. NPE cell-currents were preserved in KCNQ4 KO mouse. In conclusion, KCNQ4 may participate in the homeostasis of K+ in the subretinal space and the ciliary body, contributing to transepithelial transport.

Cellular and Molecular Neurobiology

P102.-Histone acetylation in reactive astrocytes: Is microglia the triggering "sparkle"?

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Astrocytes are essential in keeping CNS homeostasis and bringing metabolic support to neurons. It was shown that microglia activated by Lipopolysaccharide (LPS) promotes astroglial polarization to the pro-inflammatory and neurotoxic phenotype A1. We showed that this polarization is TLR4/NFκB dependent (Rosciszewski et al., 2018).In different peripheral cell types, NFκB can recruit enzymes with chromatin remodelling functions (e.g. histone acetyltransferase p300). We here aimed to understand if NFκB activation primes chromatin in A1 astrocytes through histone acetylations. We used primary cultures of glial cells obtained from C57BL/6 mice. After microglia depletion, cultures were exposed to LPS 25ng/ml for different times. NFκB activation and histone 3 acetylation (H3ac) was evaluated by immunofluorescence and immunoblotting. Astrocyte or microglia were identified as GFAP+ or IBA1+ cells respectively. Nuclear localization of NFκB subunit p65 was used as parameter of activation. Our results show that NFκB is activated in astrocytes at 1 h LPS (not before) remaining active for at least 6 h. Activation at 1 h significantly increased when microglia was added to cultures. However, we were not able to detect global changes in H3ac after LPS exposure in microglia-depleted primary cultures of astrocytes. We conclude that microglial cells may be the key to induce chromatin remodelling by facilitating NFκB activation.

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Cellular and Molecular Neurobiology

P103.-Posttraslational modification of tubulin by tyrosine analogues alters mitochondrial transport mediated by molecular motors

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