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Abstracts of the 2018 Meeting of Argentine Society for Research in Neurosciences

XXXIII Congress of the Argentine Society for Research in Neuroscience
October 24th – 26th, 2018
Pabellón Argentina, Ciudad Universitaria, UNC
Web site: http://www.saneurociencias.org.ar/

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The Argentine Society for Research in Neurosciences (SAN) held its XXXIII Annual meeting in the Argentine Pavilion (Pabellón Argentina) at the National University of Córdoba, city of Córdoba, Argentina, on October 24 and 26 of this year. The 2018 meeting took place especially under the framework of the centenary of the Córdoba University Reform of 1918.

SAN 2018 meeting had a great call with about 400 attendees among researchers, scholars, PhD students, and guests from different centers and universities of Argentina and abroad from other 11 countries of Latin America (Brazil, Uruguay, Chile, México, and Colombia), North America (USA and Canada), and Europe (Denmark, Switzerland, Ireland, and Spain). The scientific program included a total of 4 Plenary Lectures, 10 Symposia, 10 Youth Investigator Lectures, 14 Oral Communications, and 287 Posters, covering a great variety of areas in the field of neurosciences.

It is noteworthy that two of the Plenary Lectures were placed in honors of the pioneers of neurochemistry and neurobiology in Argentina, Drs. Ranwel Caputto and Eduardo De Robertis. This year the Ranwel Caputto Lecture was delivered by Prof. Charles Gilbert of Rockefeller University (USA) and De Robertis Lecture by Prof. Claudio Cuello of McGill University (Montreal, Canada). The opening lecture was delivered by Prof. Annie Andrieux (Grenoble, France), and the forth plenary lecture by Prof. Steven Fliesler of Buffalo University (USA).

As pre-meeting activities, on October 22 and 23, two specific courses were held: (a) A workshop tribute to the memory of Prof. Ricardo Miledi, pioneer in the study of synaptic transmission and ion channels, held at the Mercedes and Martin Ferreyra Institute (INIMEC CONICET, Córdoba), in which 77 undergrads and PhD students participated, as well as (b) a course entitled "Neurobiology of drug addiction," held at the School of Chemical Sciences (UNC, CONICET), which had 65 attendees and invited speakers from all around the world. In addition, on October 23, we organized a day of communication of neurosciences, open to the public, and held at the conference room of Pabellón Argentina of the National University of Córdoba.

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 176 PhD students, 61 undergrads, and 27 postdocs.

Lecture Abstracts

Wednesday, 24: 11:00–12:00 Opening Lecture/Room A

Tubulin Tyrosination-Detyrosination Cycle: Key Role in Neuronal Functions

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Microtubules are cytoskeletal polymers of α/β tubulin heterodimers, centrally involved in cell division, motility, and morphogenesis. In the de/tyrosination cycle of tubulin, the C-terminal tyrosine of α-tubulin is removed by a carboxy peptidase (TCP),

Cellular and Molecular Neurobiology

P57. Leptin-Mediated Transcriptional Regulation of Pomc in Hypothalamic Neurons

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Although it is well accepted that the adipostatic hormone leptin activates Pomc expression in hypothalamic neurons, the mechanisms controlling this interaction remain unexplored. In the brain, leptin binds to the long form of the leptin receptor stimulating the intracellular phosphorylation of STAT3 which acts as a transcription factor of several genes by acting on STAT3 binding motifs. We have detected that the neuronal Pomc enhancer 1 (nPE1) contains two canonical STAT3 binding motifs (5′-TTCCNGGAA-3′), which are highly conserved in mammals. To challenge the hypothesis that these sites participate in leptin's induced Pomc expression, we generated mutant mice lacking both STAT3 sites from nPE1 using CRISPR/Cas9 technology. To maximize leptin's effect on hypothalamic Pomc expression, we previously reduced circulating leptin levels using two different experimental strategies. Our first approach was to study the effect of refeeding on mice previously fasted for 24 hr and analyze body weight variations and hypothalamic Pomc mRNA levels. Our preliminary results indicate a greater weight loss in mice lacking STAT3 sites after fasting and a more rapid regain of previous body weight. The second approach involves crossing nPE1(STAT3-less) mice with leptin-deficient (ob/ob) mice. Further progress of these experiments will give us the possibility to evaluate the implication of STAT3 binding sites in the regulation of hypothalamic expression of POMC induced by leptin.

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P58. The Role of Sleep in the Consolidation of New Words in Temporal Lobe Epilepsy: Preliminary Results

Nerea Herrero[1], Matias Bonilla[1], Silvia Kochen[1], Luz Bavassi[2], Lucía Kaczer[2] and Cecilia Forcato[1]

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Abstract not available

Cellular and Molecular Neurobiology

P59. Impact of the Val66Met Polymorphism on the BDNF Gene on the Structure and Function of Dopaminergic Neurons

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A single nucleotide polymorphism (SNP) in the BDNF gene is present in more than 25% of the human population, and it results in a valine (Val) for methionine (Met) substitution (Val66Met) within its prodomain sequence. This SNP is associated with increased susceptibility to develop certain psychiatric and neurodegenerative disorders. Some of the associated diseases involve dopaminergic (DA) neuron dysfunction such as schizophrenia, addictions, and, in some populations, Parkinson's disease. It has been demonstrated that the Met variant of the BDNF prodomain affects hippocampal neuron structure, but its effects on DA neurons remain to be studied. We hypothesized that the Met variant of the BDNF prodomain affects DA neuron structure and function. Interestingly, we found that stimulation with the Met prodomain (but not the Val variant) induces superior cervical ganglion DA neuron death in culture. Moreover, mesencephalic DA neurons cultured from BDNF Met/Met knock-in mice displayed shorter processes as compared to the Val/Val littermates. Finally, BDNF Met/Met mice show increased spontaneous ipsilateral turns after the unilateral injection of the specific DA neurotoxin 6-hydroxydopamine, suggesting that DA neurons from this genotype are more susceptible to degenerate compared to Val/Val mice. Altogether, these results suggest a molecular explanation for the increased incidence of DA-related central nervous system disorders in Val66Met carriers.