

SAN2012 ORGANIZING COMMITTEE:

Juan Belforte

Facultad de Medicina, Universidad de Buenos Aires-Buenos Aires.

Sebastian Garcia

Universidad de Mendoza-Mendoza.

Mario Perello

Instituto Multidisciplinario de Biologia Celular-La Plata.

Victoria Pisano

Instituto de Investigación Médica Mercedes y Martín Ferreyra-Córdoba.

Patricia Setton

Instituto de Química y Fisicoquímica Biológica y Facultad de Farmacia y Bioquímica, Universidad de Buenos Aires-Buenos Aires.

Noelia Weisstaub

Facultad de Medicina, Universidad de Buenos Aires-Buenos Aires.

COURSE ORGANIZING COMMITTEE:

Jóvenes Investigadores en Neurociencias de Córdoba, (JIN) Córdoba, Argentina. **Franco Mir and Evelin Cotella**

Jóvenes Investigadores en Neurociencias de Cuyo (NeuroCuyo) Mendoza, Argentina. **Carolina Ayala**

XXVII Congreso Anual de la Sociedad Argentina de Investigación en Neurociencias 1-5 Octubre, 2012. Huerta Grande, Córdoba, ARGENTINA

SAN Course: "Sculpting the Architecture and Physiology of the Brain: Hormones have a lot to Say!". Endocrine implications for developmental programming, reproduction and behavior

COURSE PROGRAM

Day 1 – Monday, October 1st Developmental Programming

08:00-09:00 Registration

09:00-10:30 Lecture I: Principles of Neuroendocrinology. **Tony M. Plant**, Department of Obstetrics, Gynecology and Reproductive Sciences, University of Pittsburgh School of Medicine and Magee Womens Research Institute, Pittsburgh, USA. This first lecture is intended to provide students knowledge of the basic concepts underlying the field of neuroendocrinology and therefore serve as a platform for more detailed consideration to further develop specific topics throughout the

10:30-11:00 Coffee break

course.

11:00-12:30 Lecture II: Impact of steroids during development: Sexual differentiation of the brain. **María Julia Cambiasso**, Instituto de Investigación Médica Mercedes y Martín Ferreyra, INIMEC-CONICET-Universidad Nacional de Córdoba. Córdoba, Argentina

The main aim of this lecture is to examine the organizing effects of gonadal steroids on the Central Nervous System. Focus will be on the establishment of sex differences on neuron physiology and growth.

12:30-14:00 Lunch

14:00–15:30 Lecture III: Neuroendocrinology around the World. Janete
A. Anselmo-Franci, Faculdade de Odontologia, Universidade de Sao Paulo; Riberao Preto, Brasil.

Neuroendocrinology is one of the main topics in neuroscience research; the INF is in charge of the diffusion of the activities related to it all around the world. It will be interesting to know how these activities are carried on and how students are able to participate in them. Neurochemistry and Neuropharmacology Poster Number 157 / Session II

Transgenic C. elegans as a model of congenital myasthenic syndromes

Ignacio Bergé, Guillermina Hernando, Cecilia Bouzat Instituto de Investigaciones Bioquímicas Bahía Blanca. UNS-CONICET iberge@criba.edu.ar

The free living nematode Caenorhabditis elegans is a model for the study of human neurological diseases and drug testing. Our goal is to establish C. elegans as a model of slow-channel congenital myasthenic syndromes, which are originated by gain-of-function mutations in nicotinic receptor subunits. We introduced a mutation in the 9' position of the M2 domain of UNC-38 (V9'S), an essential alpha-type subunit of muscle levamisole-sensitive nicotinic receptor (L-AChR), and generated transgenic worms that express the mutant subunit in muscle. Single-channel recordings from isolated muscle cells show a dramatic increase (about 10-fold) in the open duration of L-AChR channels. Single openings appear, in contrast to wild-type channels, grouped into long activation periods. Macroscopic currents are 3-fold smaller than wild-type currents and do not decay in the presence of ACh. The functional changes of L-AChR in the mutant worm mimic those observed in vertebrate AChRs carrying the equivalent mutation. Our results reveal a high degree of conservation of functional roles of amino acids between C. elegans and human AChRs, thus opening doors for studying other gain-of-function mutations associated to slow-channel syndromes.