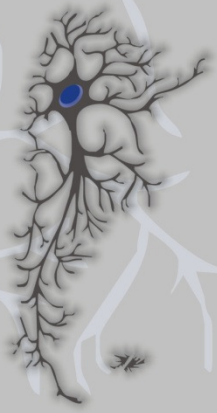
XXVIII CONGRESO ANUAL DE LA SOCIEDAD ARGENTINA DE INVESTIGACIÓN EN NEUROCIENCIAS

&

Reunión Satélite / Neurobiología del Comportamiento:
"Neuroetología y Neurobiología de la Memoria en el Cono Sur"

Septiembre 30 - Octubre 4, 2013, Huerta Grande, Córdoba, Argentina.



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SOCIEDAD ARGENTINA DE
INVESTIGACIÓN EN NEUROCIENCIAS

XXVIII CONGRESO ANUAL DE LA
SOCIEDAD ARGENTINA DE INVESTIGACION EN NEUROCIENCIAS

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Reunión satélite sobre Neurobiología del Comportamiento:
"Neuroetología y Neurobiología de la Memoria en el cono sur"
Un homenaje a Héctor Maldonado

PROGRAM

Monday September 30th: SATELLITE DAY 1

09:00: **Registration**

10:30: **Introduction**

11:00: **Symposium on Neurobiology of Memory I - International Society for Neurochemistry Symposium (**Room A)
Chair: Arturo Romano
Jorge Quillfeldt, Dep. de Biofísica, PPG Neurociências ICBS. Universidade Federal de Rio Grande do Sul, Brasil.
"Exploring the possible physiological roles of memory reconsolidation: reativation enables updating, precision-keeping and strenghtening"
Arturo Romano, IFIBYNE-CONICET, FCEN-Universidad de Buenos Aires, Argentina
"Enduring memories and the NF-kB-dependent chromatin regulation"
Rafael Pagani, Departamento de Cs Fisiológicas, FMED-Universidad de Buenos Aires, Argentina.
"Understanding Learning Disability"
Valeria Della Maggiore, Departamento de Cs Fisiológicas, FMED, Universidad de Buenos Aires, Argentina.

Neurochemistry and Neuropharmacology
Poster Number 175 / Session 1

Establishing C. elegans models of human congenital myasthenic syndromes

Ignacio Bergé, Guillermina Hernando, Cecilia Bouzat
Instituto de Investigaciones Bioquímicas de 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. In humans, gain-of-function mutations in muscle nicotinic receptor (AChR) subunits lead to slow-channel congenital myasthenic syndromes. We here explored if homologous mutations in C. elegans subunits mimic the molecular and functional changes observed in patients. In the essential UNC-38 and UNC-29 subunits of the levamisole-sensitive AChR (L-AChR) we mutated residues at position 9' of M2, which forms the gate of the channel, and position 12', which mimics a mutation found in a patient. We generated transgenic worms expressing the mutant AChRs in muscle using both wild-type and null-mutant strains as backgrounds. Electrophysiological studies show a dramatic increase (14-fold) in the open duration of L-AChR channels, and a decrease in the desensitization rate of macroscopic currents elicited by ACh, similarly to the changes detected in human mutant AChRs. Unexpectedly, no significant changes in locomotion and levamisole-sensitivity of transgenic worms occur. Overall, our results show that mutant subunits are incorporated into functional L-AChRs and lead to kinetic changes similar to those observed in vertebrate AChRs, thus revealing a high degree of conservation of functional roles of amino acids between C. elegans and human AChRs. These results open doors for establishing C. elegans models for human myasthenic syndromes.