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PRE-CONGRESS COURSE "NEUROBIOLOGY OF DRUG ADDICTION"

SAN IBRO LARC Course and ISN Small Conference (ISN-CC) Associated to the XXXIII SAN 2018 Meeting

October 22nd -23rd, 2018

Ciudad Universitaria, Córdoba, Argentina

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-Salón Auditorio, Edificio Integrador, Facultad de Ciencias Químicas, Universidad Nacional de Córdoba.
-Salón de Actos Pabellón Argentina, Ciudad Universitaria, Córdoba, Argentina

WORKSHOP *Homage to Ricardo Miledi*
"Workshop: Past, Present and Beyond of Synaptic Transmission"

Previous and satellite activity of the XXXIII Annual Congress of the Argentine Society of Neuroscience Research – SAN

October 22th – 23th, 2018 – Instituto Martín y Mercedes Ferreyra, Córdoba

LOCATION:
Instituto de Investigaciones Médicas
Mercedes y Martín Ferreyra (INIMEC)
Ciudad de Córdoba, República Argentina

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P238-Phosphorylation of intracellular tyrosines modulates the ionotropic function of the α7 nicotinic receptor

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α7 is expressed in the brain and contributes to cognition, attention, and memory. It contains an extracellular domain with the agonist binding sites; a transmembrane domain, which forms the ion pore; and an intracellular domain (ICD), which contains sites for modulation and intracellular signaling. The mechanisms by which the cell can regulate the ionotropic function of α7 remain unknown. We explored how intracellular phosphorylation affects α7 activity by patch clamp recordings in HEK cells expressing α7. Wild-type α7 channel activity elicited by ACh appears as brief isolated openings and as activation episodes containing a few brief openings in quick succession (bursts). Preincubation of cells expressing α7 with the inhibitor of Src family kinases (PP2) increased significantly the mean burst duration. The exposure of cells to PP2 during the course of the recording revealed a significant increase in the frequency of channel opening in addition to the increase of burst durations. To confirm that these changes were due to the inhibition of phosphorylation of α7-ICD, we introduced mutations at potential phosphorylation sites (Y386F and Y442F). The mutations prolonged burst durations, thus mimicking the effects of PP2. Also, the mutants were insensitive to PP2, confirming that Y386 and Y442 are responsible for its effects on α7 kinetics. Our results indicate that dephosphorylation positively modulates α7 channel activity in a way compatible with decreased desensitization.