SAN2021
EBOOK

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PROGRAM

	OCT 18	OCT 19	OCT 20	OCT 21	OCT 22
09:00 11:00		**SYMPOSIA** Tue-S1 to Tue-S3: Alba, Bisig, Biurrun&Coronel	**OC SESSIONS** (OC1- OC4)	**SYMPOSIA** Thu-S7 to Thu-S9: Kaczer, Pallares, Setton	**YOUNG INVESTIGATORS TALKS** Fri-YIT4 to Fri-YIT5
11:00 11:30	**Opening Words** (Cancela) "EDUARDO DE ROBERTIS" **LECTURE** **Rita Raisman-Vozari** (Chairs: Ferrario, Antonelli, Stahl)	Biobreak			**E-SOCIAL** **Scientific publications: Journals and editorial policies** (Rayes, Zorrila, Ceriani) **Gender inequities and inequalities around the world** (Antonelli, Murta)
11:30 12:30		**PLENARY LECTURE** **Peter Kalivas** (Chairs: Cancela, Pacchioni, Pautassi, Coll)	**PLENARY LECTURE** **Andrea Nistri** (Mazzone, Unsain)	**PLENARY LECTURE** **Maria Dolores Ledesma** (Sodero, Adamo)	
12:30 13:00			Biobreak		
13:00 14:00			**E-SOCIAL** **Co-authorship network structure and gender inequalities of the Argentine neuroscientific community** (Bekinstein, Fernandez)		
14:00 16:00	**E-POSTER SESSIONS** (PS1 - PS3)	**E-POSTER SESSIONS** (PS4 - PS7)	**SYMPOSIA** Wed-S4 to Wed-S6: Rela, Echeveste&Samengo, Wilson&Moyano	**E-POSTER SESSIONS** (PS8 - PS10)	**SYMPOSIA** Fri-S10 to Fri-S11: Durand, Monteleone&Brocco
16:00 16:30			Biobreak		
16:30 17:30	**YOUNG INVESTIGATORS TALKS** Mon-YIT1 to Mon-YIT3	**PLENARY LECTURE** **Silvia Bunge** (Andreau, Brocco)	**PLENARY LECTURE** **Vivian Budnik** (Rayes, Contin)	**PLENARY LECTURE** **M. Laura Feltri** (Setton, López)	"RANWELL CAPUTTO" LECTURE **Carlos Dotti** (Chairs: Cancela, Guido, Sodero, Fernández)
17:30 18:00		Biobreak			
18:00 19:30	**E-SOCIAL** **Navigating the gray areas to do Neuroscience** (Mazzone, Rayes)	**E-SOCIAL** **Socio-environmental modulation of cognitive processes** (Fernandez Larrosa, Andreau)	**E-SOCIAL** **Looking for training abroad? Tips for international interviews** (Zorrilla, Beckwith, Fernandez)	**ASAMBLEA SAN** Elecciones	**E-SOCIAL** **Neuro-cine** (Ferrario, Avale)

uncleavable signal peptide giving this receptor special trafficking and signaling features. Our aim is to characterize UCNs/CRHR2α signaling pathways in a neuronal context using the hippocampal cell line HT22 stably expressing the receptor (HT22-CRHR2α) and primary neuronal cultures. ERK1/2, CREB and Akt were activated downstream CRHR2α in HT22-CRHR2α cells. ERK1/2 and CREB activation depended on cAMP generated by the soluble adenylyl cyclase (sAC) and transmembrane adenylyl cyclases but only sAC was required for Akt activation. Upon stimulation, HT22-CRHR2α cells undergo morphological changes that required sAC-produced cAMP and PKA activation independently of ERK1/2 activation. We observed an unusual trafficking for the CRHR2α due to the pseudo-signal peptide: the fraction of the receptor in the cell surface increased 6 min after stimulation returning to basal levels after 30 min. In primary neurons, preliminary results suggest that Fos induction by the CRH system may depend on neuronal activation and the ligand used. Our results highlight the relevance of cellular contexts and provide information to define the role of UCNs and CRHR2α in the CRH system.

A Functional Interaction Between a Region of the SARS-CoV-2 Spike Protein and the Human α7 Nicotinic Receptor

Juán Facundo Chrestia

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Coronavirus disease 2019 (COVID-19) is caused by the severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2). The binding of the viral spike protein (S) to angiotensin-converting enzyme 2 in host cells is crucial for infection. The S protein has been suggested to interact with nicotinic acetylcholine receptors (nAChRs), and their contribution to the COVID-19 inflammatory pathophysiology has been proposed. α7 is an interesting candidate target because it is present in neuronal and non-neuronal cells, and it has neuroprotective and anti-inflammatory actions. By whole-cell and single-channel recordings we revealed that the Y674-R685 region of the S protein shows a direct functional interaction with human α7 nAChR. The S fragment exerts a dual effect, acting as a low-efficacy agonist and a non-competitive antagonist. In agreement with molecular dynamics simulations showing stable binding of this region to the ACh binding pocket, the S fragment activates α7, but only in the presence of a potentiator, supporting its action as a very low-efficacy agonist. In addition, it allosterically inhibits α7 responses elicited by ACh, which may result in the predominant effect. This study provides unequivocal evidence supporting a functional α7-S protein interaction, which may play a role in infectivity and/or disease progression and may be explored for new therapeutic opportunities.

Effects of the antipsychotic drug chlorpromazine on D1/D5R constitutive activity and postsynaptic currents in prefrontal cortex (PFC) neurons

Clara Inés McCarthy

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The PFC is a key associative cortical region that is severely affected in patients with schizophrenia (SCZ). Changes in dopamine receptor type 1 (D1R) function and availability in the PFC are associated with working memory deficits of SCZ. We previously showed that D1R constitutive activity increases voltage-gated calcium channels CaV2.2 density in the cell surface in transfected cells and that this effect is relevant in the PFC. Here, we continue to study D1/D5R constitutive activity modulation of native CaV currents and explore its impact on synaptic activity in PFC pyramidal neurons. We performed patch-clamp experiments and cAMP measurements on transfected HEK293T cells and wild-type C57BL/6 mouse brain slices, combining two pharmacological interventions to discriminate D1/D5R activity-dependent effects: systemic administration of chlorpromazine (CPZ, D1/D5R inverse agonist) and intra-PFC infusions of SCH23390 (D1/D5R antagonist). We assessed CaV subtype contributions to total native calcium current in naïve and treated mice. Then, we evaluated the impact of this pharmacological manipulation on synaptic activity: we recorded evoked, spontaneous and miniature excitatory/inhibitory postsynaptic currents (E/IPSC). We found that CPZ-treatment decreased EPSCs while increasing IPSCs, creating an E/I imbalance that favored inhibition. We are conducting experiments to further understand the link between D1/D5R constitutive activity and the changes seen in postsynaptic currents.