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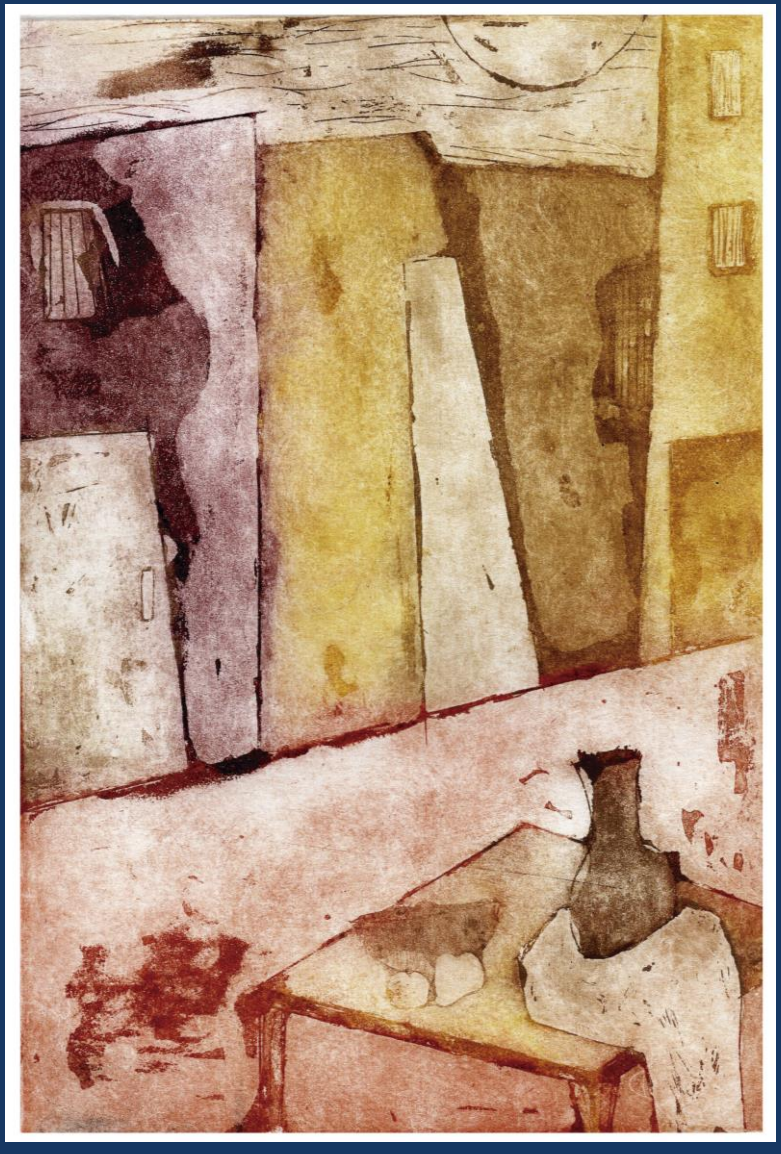

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La Tapa (Ver pág. 4)
Atardecer en la tarde
Antonella Ricagni

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REUNIÓN ANUAL DE SOCIEDADES DE BIOCIENCIA 2019

LXIV Reunión Anual de la
Sociedad Argentina de Investigación Clínica (SAIC)

LI Reunión Anual de la
Asociación Argentina de Farmacología Experimental (SAFE)

XXI Reunión Anual de la
Sociedad Argentina de Biología (SAB)

XXXI Reunión Anual de la
Sociedad Argentina de Protozoología (SAP)

IX Reunión Anual de la
Asociación Argentina de Nanomedicinas
(NANOMED-ar)

VI Reunión Científica Regional de la Asociación Argentina de Ciencia y Tecnología de Animales de Laboratorio (AACyTAL)

con la participación de
The Histochemical Society

13 - 16 de noviembre de 2019
Hotel 13 de Julio - Mar del Plata

EDITORES RESPONSABLES

Dra. Mónica Costas
Dra. Gabriela Marino
Dr. Pablo Azurmendi

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ANNUAL MEETING OF BIOSCIENCE SOCIETIES 2019

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with the participation of
The Histochemical Society

November 13th – 16th, 2019
Hotel 13 de Julio - Mar del Plata

CHIEF EDITORS

Dra. Mónica Costas
Dra. Gabriela Marino
Dr. Pablo Azurmendi

(p<0.05 vs. HypoT). But in EuT hearts perfused Amd did not induce postischemic changes. In both types of administration, Amd increased LVEDP in EuT and HypoT hearts. Summing up a) Oral Amd prevents severe I/R dysfunction only in EuT hearts, not adding more protection to HypoT ones; b) Amd directly perfused only evidenced its Ca^{2+} channels inhibition; c) Cardioprotection of oral Amd could be due to induction of a "cardiac hypothyroid effect".
Supported by UNLP- X795 grant.

0901 - SEX DIFFERENCES IN BLOOD PRESSURE RESPONSE TO CONTINUOUS ANG II INFUSION: ARE SEX HORMONES THE ONLY ONES TO BLAME FOR SUCH DIFFERENCES?

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INSTITUTO DE INVESTIGACIÓN MEDICA MERCEDES Y MARTÍN FERREYRA - INIMEC-CONICET-UNC (1); CÁTEDRA DE FISIOLOGÍA ANIMAL. FCEFYN - UNIVERSIDAD NACIONAL DE CÓRDOBA (2)

Abstract/Resumen: Evidence demonstrate that the pressor response to Ang II infusion is sexually dimorphic under physiological and pathophysiological circumstances. But why do male and female show differences in rennin angiotensin system (RAS) activation and inhibition? Sex steroids can induce organizational (long-lasting or permanent) effect during critical periods of development but can also impart (temporary or reversible) activational effects. Furthermore, males and females also carry different sex chromosome complements (SCC:XY/XX) and thus are influenced throughout life by different genomes. Previous evidence demonstrates a modulatory effect of SCC in RAS receptor expression (brain and renal), as well as in the Ang II sexually dimorphic bradycardic barroreflex and hypertensive responses. In the present study we evaluated the involvement of SCC, organizational and activational hormone effect on changes in mean arterial pressure (MAP) in a 10 min Ang II infusion protocol. For this purpose, we used gonadectomized (Gdx) mice of the "four core genotype" model, in which the effect of gonadal sex and SCC is dissociated, allowing comparisons of sexually dimorphic traits between XX and XY females as well as in XX and XY males. For hormonal replacement experiments Gdx mice were daily injected with ß-estradiol or testosterone propionate (2 ug/g) for a 4 day period. The statistical analysis reveals an interaction of SCC, organizational and activational hormonal effect during Ang II infusion {F (7.39=2.60, p<0.01)}. Our results indicate that in absence of the activational hormonal effects an interaction between the SCC and the organizational hormonal action differently modulate changes in the arterial pressure. Furthermore, estrogen and testosterone exert important activational effects on changes in MAP during Ang II acute continous infusion. Thus, our data demonstrate the contribution and interaction of SCC, activational and organizational hormonal effects in sex differences in blood pressure regulation.

Neurociencias / Neurosciences I

Chairs: Fernando Correa | Flavia Saravia

0027 - INSULIN RECEPTOR ACTIVATION EFFECTS ON SYNAPTOSOMAL 2-AG HYDROLYSIS IN AN AMYLOIDOSIS MODEL INDUCED BY Aß OLIGOMERS

Ana Clara PASCUAL | Sabrina Rosicler SALAS | Susana Juana PASQUARE

INIBIBB-CONICET, DEPTO. BIOLOGÍA, BIOQUÍMICA Y FARMACIA-UNS

Abstract/Resumen: Insulin (Ins) plays an important role in synaptic plasticity and is tightly related to Alzheimer´s disease (AD). Aß oligomers (OAß), which are responsible for synaptic dysfunction in AD, can bind to Ins receptor (IR) and can therefore be internalized into neurons. OAß also disrupt the synaptic membrane and diminish 2-AG availability, the main neuroprotective cannabinoid. Ins can prevent OAß binding to IR, thus attenuating its neurotoxicity. Here, we hypothesized that Ins prevent OAß deleterious effects on 2-AG metabolism. To this end, we isolated cerebral cortex synaptosomes (syn) by differential centrifugation purified in ficoll gradients and preincubated them with 10 µM LY294002 (phosphatidylinositol-3-kinase -PI3K- inhibitor) or 100 µM genistein (tyrosine kinase inhibitor) for 10 min, and subsequently incubated with 0.2 mM vanadate (protein-tyrosine phosphatase inhibitor), 100 nM Ins, or 0.2 mM vanadate plus 100 nM Ins, for 30 min. Syn were then incubated for 10 min with or without 0.1 µM OAß. After this incubation, activation of IR signaling by Western blot, released LDH activity, and 2-AG hydrolysis activity were evaluated. It was observed that a 30 min incubation with Ins and vanadate activated IR and Akt (p<0.05). The subsequent incubation with OAß did not alter IR activation (p>0.05). As to syn membrane damage, neither of the pretreatments could prevent OAß effect on LDH release (p>0.05). On the other hand, Ins and vanadate decreased 2-AG hydrolysis (p<0.01) and their effect was not observed if syn were preincubated with LY (p>0.05). However, in the presence of OAß, Ins and vanadate failed to alter 2-AG hydrolysis (p>0.05) and LY increased this activity (p<0.001). Our results show a regulation of 2-AG hydrolysis by Ins, possibly increasing its availability via IR and involving PI3K pathway, which is abolished by OAß. The effect of OAß appears to be independent of IR and to involve PI3K activity. Ins also failed in preventing OAß damage in synaptic membrane.

0037 - SPINAL CORD INJURY DRIVES CHRONIC HIPPOCAMPAL CHANGES

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INSTITUTO DE BIOLOGÍA Y MEDICINA EXPERIMENTAL (IBYME-CONICET) (1); ACHUCARRO BASQUE CENTER FOR NEUROSCIENCE (2)

Abstract/Resumen: After spinal cord injury (SCI), patients exhibit cognitive deficits that could be related to hippocampal alterations. The objectives of this work were:1) to determine which step in the neurogenic process was altered after chronic SCI; 2) to explore the role of acute glucocorticoids (GC) and transneuronal degeneration in chronic neurogenesis reduction after SCI; 3) To evaluate cognitive hippocampal dependent-tasks after chronic SCI. In order to perform the first objective, we used Nestin-GFP mice combined with multiple immunolabeling (BrdU, GFAP, doublecortin DCX) and confocal microscopy. Survival and mitotic capability of neural stem cells (NSCs, Nestin-GFP+/GFAP+) and amplifying progenitors (ANP, Nestin-GFP+/GFAP-) were assessed by labeling these cells with BrdU. The number of DCX + cells together with mitotic NSCs and ANPs was downregulated after 60 days post-injury (dpi) (p<0.05, SCI vs. sham). To comply with the second objective, GC action was blocked using the GC receptor antagonist, RU-486 during the acute phase and neurogenesis was measured 60dpi. This result implied acute GC in chronic neurogenesis reduction since the number of DCX + cells was restored after RU-486-treatment (p<0.01, SCI vs. SCI+RU486). On the other hand, spinal cord hemisection was performed in order to lacerate axons of only one side. Afterwards, neurogenesis 60 dpi was measured in the ipsilateral and contralateral hippocampus. Neurogenesis decreased in the contralateral side with respect to the ipsilateral side (p<0.05), which would involve transneuronal degeneration in this downregulation. To achieve the last objective, cognitive hippocampal dependent-tasks were evaluated using the novel object recognition and Y-maze test. After SCI, animals showed deficits in recognition (p<0.01, SCI vs. Sham) and spatial working memory (p<0.01, SCI vs. sham) 60 dpi. These results support that acute GC and transneuronal degeneration caused