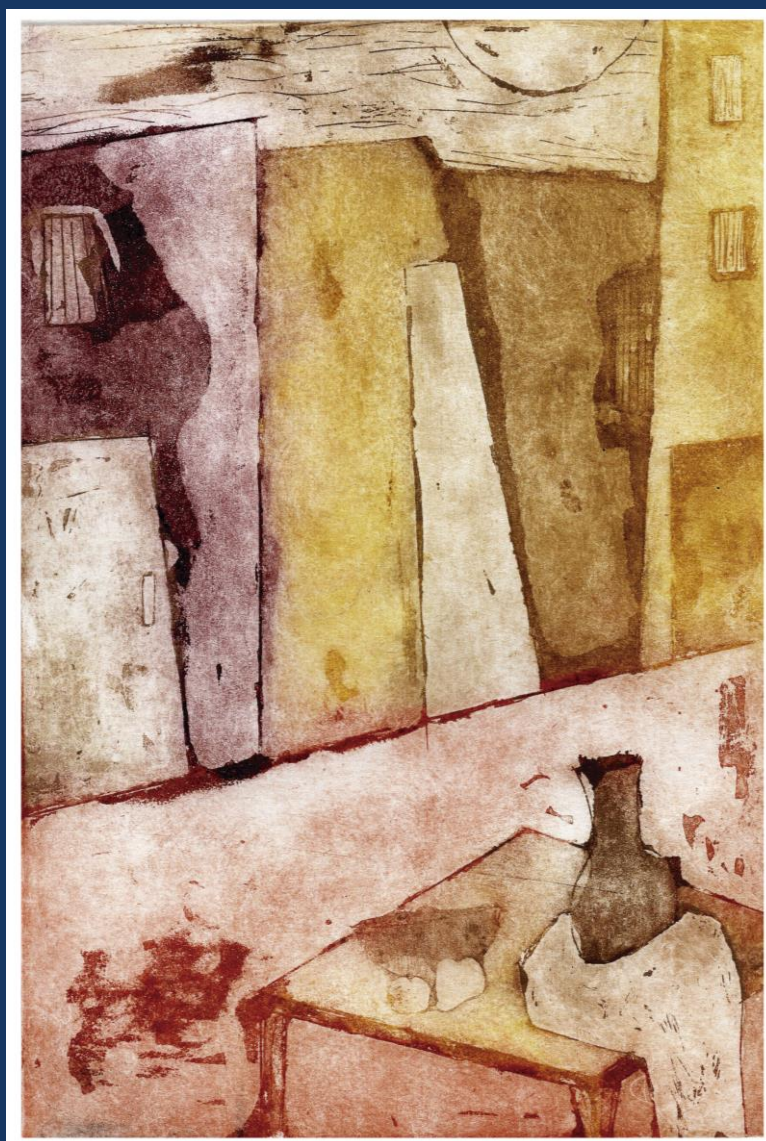


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REUNIÓN ANUAL DE SOCIEDADES DE BIOCIENCIA 2019

**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

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**Dra. Mónica Costas
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Dr. Pablo Azurmendi**

ANNUAL MEETING OF BIOSCIENCE SOCIETIES 2019

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CHIEF EDITORS

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

estrogen receptor: ERalpha or GPR30. cPKC activation precedes that of ERalpha. cPKC probably uses intermediaries to phosphorylate ERalpha. Several kinases can be phosphorylated by cPKC and are able to phosphorylate the ERalpha, among them we find GSK3β. The aim of this study was to evaluate the role of GSK3β in the E17G-induced alteration of Mrp2 activity. IRHC were treated with GSK3β inhibitors Li (3 mM) or BIO (1 μM) and then exposed to E17G (100 μM). To investigate in which pathway GSK3β participates, IRHC were exposed to BIO and inhibitors of ERalpha (ICI182,780, ICI, 1 μM), cPKC (Gö6976, Gö, 1 μM) or PI3K (Wortmannin, W, 100 nM) before exposure to E17G. All preparations were incubated with CMFDA (intracellularly converted in glutathion-methylfluorescein [GMF], substrate of Mrp2). IRHC accumulating GMF in their canalicular vacuoles (cVA) were counted and compared to control IHRC. Results (% Control): GSK3β inhibition (Li+E17G: 71 ± 7b; BIO+E17G: 70 ± 5b) partially prevented the effect of E17G (48 ± 4a) on cVA of GMF. The preventive effects of W (W+E17G: 75 ± 5b) and BIO on the decrease in cVA induced by E17G were additive (BIO+W+E17G: 91 ± 1a,c). Contrarily, the preventive effects of ICI (ICI+E17G: 69 ± 3b) or Gö (Gö+E17G: 77 ± 3b) did not modified BIO protective effects (BIO+ICI+E17G: 72 ± 2b) and (BIO+Gö+E17G: 77 ± 10b). a: significantly different from Control; b: significantly different from E17G and Control; c: significantly different from E17G+BIO and E17G+W. BIO, Li, W, Gö, and ICI did not affect % cVA. (p<0.05, n= 3). GSK3β inhibition protects against E17G-induced impairment of Mrp2 transport, indicating a role of the kinase in estrogen cholestasis. Co-inhibition studies suggest that GSK3β participates in the same pathway of ERalpha and cPKC and in different pathway of PI3K (downstream of GPR30).

0875 - ATRIAL NATRIURETIC PEPTIDE (ANP) ENHANCES ANTIOXIDANT CAPACITY IN EXPERIMENTAL ACUTE PANCREATITIS

Ana Paula COURREGES (1) | Guadalupe ALVAREZ(1) | Mario CONTIN(2) | Federico OCHOA(3) | Fabiana LAIRION(4) | Marisa REPETTO(4) | Marcelo VATTA(5) | Liliana G. BIANCIOTTI(1)

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Abstract/Resumen: We previously reported ANP attenuates the severity of acute pancreatitis by reducing trypsinogen activation and the inflammatory response. Recent studies support that endoplasmic reticulum (ER) stress and oxidative stress (OS) precede these events. Indeed, we showed that ANP attenuates ER stress and stimulates ER-dependent apoptosis. ER stress is intimately related to OS in the pathophysiology of numerous diseases. Given that the exocrine pancreas is rather susceptible to OS due to the extremely weak expression of antioxidant enzymes, in the present we sought to establish whether ANP affected OS in experimental AP by studying the main antioxidant (enzymatic and non-enzymatic) defense. AP was induced in Sprague-Dawley strain rats (200-220 g) by four repetitive cerulein injections (40 μg/Kg). Thirty minutes before the first cerulein injection animals were infused with either saline (control) or ANP (1 μg/Kg/h) for 60 min. Following euthanasia (60 min after the last cerulein injection) pancreatic samples were harvested for further assays (CICUAL-FFYB #4107/18). ANOVA followed by a Student's t test modified by Bonferroni was used for statistical analysis. Results are expressed as the means±S.E.M. and p values of 0.05 or less were considered statistically significant. AP induces OS as previously reported. ANP stimulated Nrf-2 nuclear translocation (assessed by immunohistochemistry) which is a transcription factor that induces the expression of antioxidant enzymes (p<0.001). ANP also enhanced the activity of superoxide dismutase (SOD) (p<0.05), catalase (p<0.01) and glutathione transferase. Furthermore it also restored reduced glutathione and total

glutathione levels (assessed by HPLC- tandem mass spectrometry) to control values (p<0.05). Present findings show that ANP enhances the antioxidant defense capacity of the exocrine pancreas in AP, further supporting its beneficial role in the disease.

Neurociencias / Neurosciences III

Chairs: Claudia Bregonzio | Analía Reinés

0324 - EFFECT OF LITHIUM IN PYRAMIDAL NEURONS OF CORNUS AMMONIS

Georgina Paula OSSANI (1) | Ana Margarita UCEDA(2) | Osvaldo Juan PONZO(3) | Néstor Rubén LAGO(4) | Miguel RIUDAVETS(5) | Diego Javier MARTINO(6)

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Abstract/Resumen: Lithium (Li) is a first-line drug for long-term prophylactic treatment of bipolar disorder (BD). However, mechanisms by which lithium exerts its mood-stabilizing effects are not very clear. A decrease in the overall volume of the hippocampus (H) by imaging studies has been described in patients with BD, it has also been reported that treatment with Li would reverse this effect, highlighting its neuroprotective effect. The aim of this work was to evaluate the effect of Li on pyramidal neurons within Cornu Ammonis (CA) subregions of the H. Wistar male rats (n= 16) were randomized into two groups: control group (CG) fed ad libitum powered standard diet and experimental group (EG) fed ad libitum the same diet supplemented with 60 mmol of lithium/kg diet for 1 month. Lithium serum levels were measured and reached therapeutic values in EG (0.57 ± 0.18 mmol/L). The brains were removed for histopathological analysis, fixed, and cut coronally. From each brain we selected a section (Bregma -2.8 mm) and stained with cresyl violet. First, we took serial pictures of the entire CA region with a 60x objective starting at the midline (CA1-2-3). Serial photos were divided into 4 groups, and the first 5 photos from each of them were selected for the analysis. Then, using the Image J Software we measured the area of the cell body and nucleus of CA pyramidal neurons on each selected picture. The criteria for selecting neurons to be measured included a well-defined nucleus and nucleolus. All assessments were performed blinded to Li treatment. We observed that the mean size (μm²) of the neuronal soma and nucleus of pyramidal neurons in the third group were significantly larger: CG= 140 ± 24 vs. EG= 174 ± 36, t= -2.15, p= 0.049 for cytoplasm; and CG= 75 ± 12 vs. EG= 92 ± 16; t= -2.28, p= 0.038 for nuclear size. This sub-region could correspond to CA2 subfield. Our results support the theory that lithium acts at the H level producing an increase of the cell and nuclear area of the pyramidal neurons in a specific sub-region of the CA.

0337 - EVIDENCE OF CANNABINOID MODULATION IN NUCLEAR SIGNALING

Virginia Lucía GAVEGLIO | Norma María GIUSTO | Susana Juana PASQUARE

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