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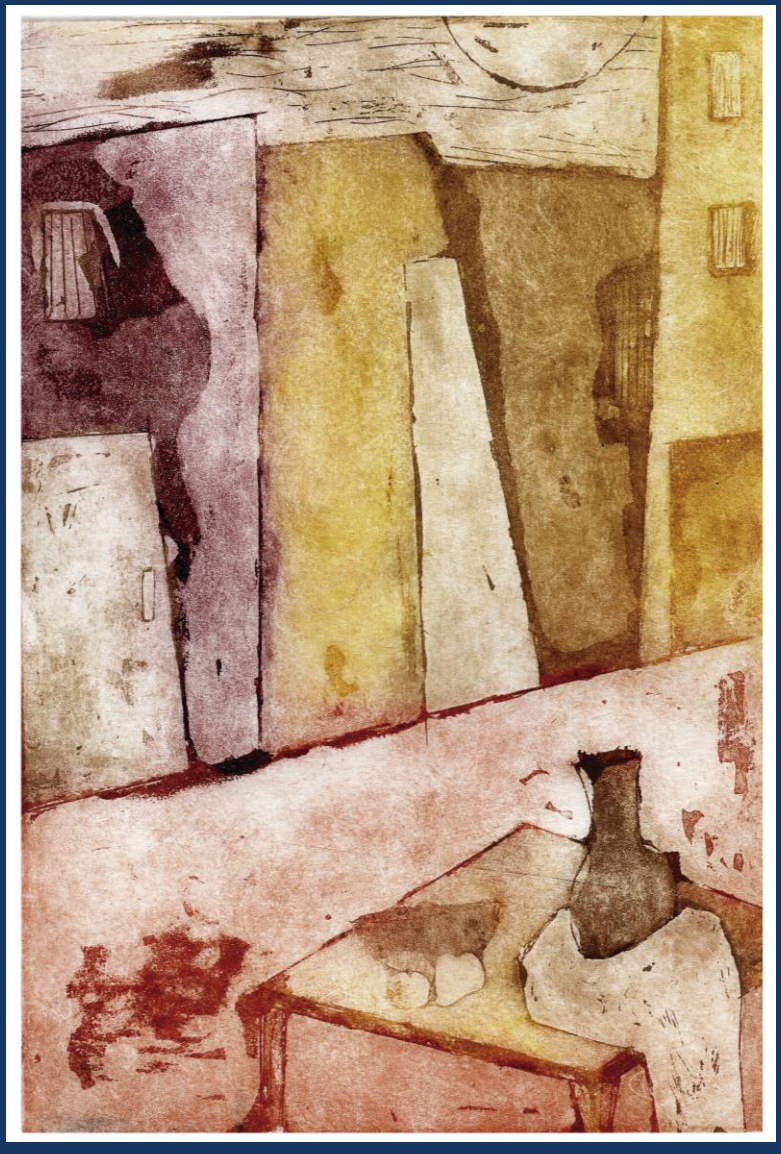

MEDICINA

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medicina

BUENOS AIRES, VOL. 79 Supl. IV - 2019

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

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Secretaría de Redacción: Ethel Di Vita, Instituto de Investigaciones Médicas Alfredo Lanari, Combatientes de Malvinas 3150, 1427 Buenos Aires, Argentina
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medicina
BUENOS AIRES, VOL 79 Supl. IV - 2019

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

medicina
BUENOS AIRES, VOL 79 Supl. IV - 2019

ANNUAL MEETING OF BIOSCIENCE SOCIETIES 2019

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CHIEF EDITORS

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

indicating cell enlargement, regardless of DEN treatment. Proliferating cell nuclear antigen (PCNA) was determined by immunohistochemistry to assess hepatocellular proliferation. GH-treated groups exhibited a non-significant increase of PCNA positive nuclei. Increased cell proliferation was also observed inside dysplastic foci, although differences were significantly only for animals that did not receive GH treatment ($p<0.05$). Therefore, similar to that observed in growing mice, prolonged GH-administration to adult mice per se does not promote tumor formation, nor is it fostered by tumor inductor adjuvant treatment.

0585 - BENZOPHENONES 2 (BP2) AND 3 (BP3) AFFECT CELLULAR ADAPTIVE RESPONSES IN THE PANCREATIC BETA CELL LINE MIN6B1 IN THE PRESENCE OF THE AUTOPHAGY INHIBITOR CHLOROQUINE

Florencia SZULAK | Marina Olga FERNANDEZ | Damasia BECU DE VILLALOBOS | **Eleonora M. SORIANELLO**

IBYME-CONICET

Benzophenones used as ultraviolet light blockers in plastic packaging of food and in sunscreens, are considered endocrine disruptors. In addition, autophagy is a mechanism of degradation and recycling of cellular components essential for cell homeostasis. In pancreatic beta cells autophagy has a fundamental role in relieving endoplasmic reticulum (ER) stress caused by misfolded proteins, including insulin. Our research focused on studying the effect of BP2 and BP3 on mouse pancreatic beta cell line MIN6B1 function in the presence of the autophagy inhibitor Chloroquine (CQ). The results showed that basal insulin secretion was inhibited by the lysosomotropic compound CQ (10 µM), and also by BP3 (10-5 M) both when incubated alone and in the presence of CQ. In addition, CQ triggered an adaptive response involving induction of genes related to lysosomal biogenesis, Lamp2, or autophagy, Sqstm1/p62. Interestingly, BP3 significantly reverted the induction of Lamp2 and showed a strong tendency to counteract the induction of Sqstm1/p62 by CQ, in addition to decreasing the mRNA levels of the autophagy marker Ulk1 both basally and in the presence of CQ. BP2 (10-5 M) only reverted the induction of Lamp2. Interestingly, these effects failed to alter the protein levels of LC3II or SQSTM1/p62, or the autophagic flux itself. Regarding ER stress markers, BP3 decreased the transcription of Xbp1 and its spliced form, and counteracted the induction of Chop and Grp78/Bip triggered by CQ. Likewise, BP2 partially reverted the induction of Grp78/BiP mRNA by CQ. We conclude that benzophenones, mainly BP3, and to a lesser extent BP2, counteract adaptive responses related to autophagy, lysosomal biogenesis and reticulum stress, in a condition of lysosomal stress and autophagy block caused by CQ. Since BP3 also inhibited basal insulin secretion, we suggest that both BP2 and BP3 alter the function of the pancreatic beta cell.

Supported by CONICET, ANPCyT, Fund. Rene Baron and Fund. Williams grants.

0642 - BLOCKING GABAB RECEPTORS (GABABR) FROM BIRTH TO WEANING INDUCES PROFOUND CHANGES IN THE GONADOTROPIC AXIS IN ADULT MICE

Marianne BIZZOZZERO HIRIART | Noelia DI GIORGIO | Esteban REPETTO | Carlos LIBERTUN | Victoria LUX-LANTOS

INSTITUTO DE BIOLOGÍA Y MEDICINA EXPERIMENTAL (IBYME-CONICET), LABORATORIO DE NEUROENDOCRINOLOGÍA

We have previously shown that administration of GABAB antagonist (CGP55845) to neonatal mice from postnatal day (PND) 2 to weaning (PND21) significantly decreased arcuate nucleus (ARC) kisspeptin (Kiss1) expression and increased dynorphin (Pdyn) expression in both sexes on PND21. Here our aim was to evaluate the effect of this sustained inhibition of GABABR signaling on the gonadotropic axis in adulthood. Neonatal Balb/c males (M) and females (F) were injected with CGP55845 (1 mg/kg, s.c., CGP) or saline from PND2-21, three times/day (8 AM, 1 PM, 6 PM) and sacrificed in adulthood. Serum samples and gonads were collected for hormonal measurements (RIA). Brains were frozen and 500 µm slices were obtained on a cryostat. ARC and anteroventral periventricular nucleus (AVPV) micropunches were obtained. Kiss1, Pdyn, neurokinin B (Tac2), tyrosine hydrolxylase (Th), progesterone receptor (Pgr) and GnRH (Gnrh1) mRNA expression was assessed in the micropunches by qPCR. Body weight (BW) and AGI (anogenital distance/BW) were evaluated on PND 7, 14 and 21. Puberty onset was determined by vaginal opening (VO) or preputial separation (PS). CGP significantly increased BW on PND21 in F ($p<0.02$) while BW was decreased by CGP at all ages studied in M ($p<0.001$). CGP significantly decreased AGI in both sexes (F: $p<0.001$; M: $p<0.02$). CGP decreased Kiss1 ($p<0.01$), Tac2 ($p<0.02$), Pdyn ($p<0.04$) and Pgr ($p<0.01$) in ARC of both sexes. In AVPV from CGP-treated F, Th was significantly decreased ($p<0.01$) and a near significant decrease in Pgr was observed ($p<0.07$). FSH was increased in CGP-treated M ($p<0.04$). In addition, CGP increased gonad progesterone ($p<0.01$) and testosterone ($p<0.002$) in F. These results demonstrate that sustained inhibition of GABABR signaling during a critical postnatal period of development and maturation of the gonadotropic axis profoundly alters many parameters of this axis in adulthood. Supported by: CONICET, ANPCYT, UBA, Fund. René Barón and Fund. Williams.

0676 - PRENATAL D-AMPHETAMINE EXPOSURE INFLUENCES PROLACTIN SYNTHESIS AND SECRETION IN RESPONSE TO STRESS AND ESTROGEN IN ADULTHOOD.

Florencia Eleonora SANTONJA(1) | Elisa PIETROBON(1) | Flavia NEIRA(1) | María Belén SÁNCHEZ(1) | Tamara MORENO SOSA(1) | Graciela Alma JAHN(1) | Susana Ruth VALDEZ(1) | Claudia BREGONZIO(2) | **Marta SOAJE** (1)

INSTITUTO DE MEDICINA Y BIOLOGÍA EXPERIMENTAL DE CUYO (IMBECU) (1); FACULTAD DE CIENCIAS QUÍMICAS, UNC, DEPARTAMENTO DE FARMACOLOGÍA, IFEC-CONICET (2)

The dopaminergic system, a main regulator of prolactin (PRL) secretion, is closely involved in the responses to stress and amphetamine. The ovarian steroids modulate PRL secretion and several aspects of stress responses. Previously, we found that prenatal exposure to amphetamine (PEA) induced changes in pituitary mRNA D2R expression during adulthood. Our present aim was to study PEA effects and the influence of estrogen (E2) on pituitary D2R expression (protein) and their correlation with pituitary PRL content (mRNA and protein levels) in adult OVX rats in response to stress. Moreover, we also explored the role of E2 in response to stress on the expression of de pTH-Ser 40 in the medium basal hypothalamus (MBH) of OVX adult rats in PEA animals. For these purposes, female Wistar rats were treated daily with D-amphetamine 2.5 mg/kg i.p./saline during days 15 to 21 of pregnancy. Their female offspring were OVX at day 60; 15 days later treated with estrogen/oil (E2; 2 x 5 µg/rat/24 h) and exposed to immobilization stress during 30 min. Blood and tissue samples were collected for corticosterone by RIA and pituitary D2R and PRL content by real time PCR and Western blot (WB) determinations. pTHSer-40 expression was determined by WB in MBH extracts. Data were analyzed using two-way ANOVA and Student´s-t test. Pituitary D2R expression (protein) was diminished only in PEA OVX+E2 rats ($p<0.05$) and no effects of stress were observed. E2 increased PRL mRNA levels in control and PEA OVX rats ($p<0.05$) and prevented the effect of stress. Stress diminished PRL pituitary content (protein) in control rats with E2 and the PRL protein in PEA rats independent of E2 treatment ($p<0.05$). Moreover, stress decreased MBH p-TH in PEA OVX+E2 rats ($p<0.05$). Thus, prenatal treatment with D-amphetamine sensitizes the hypothalamus-pituitary system affecting PRL synthesis and secretion in response to stress and E2.