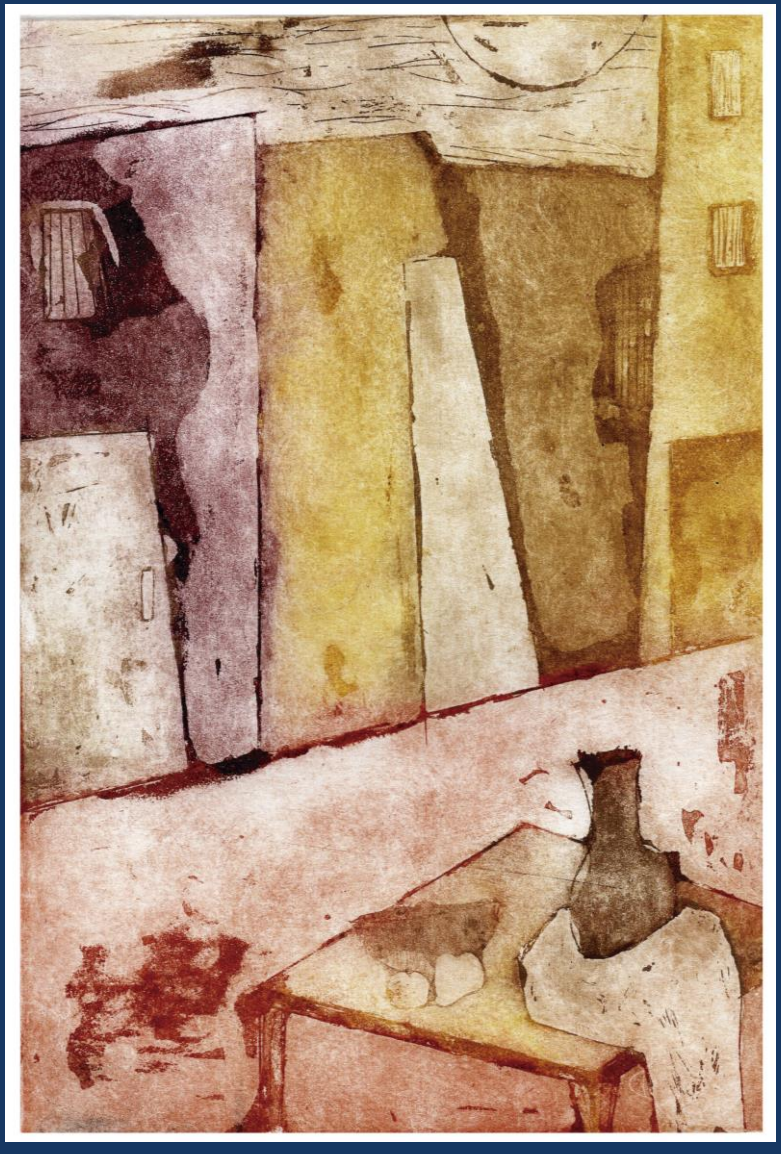
2019

medicina

BUENOS AIRES VOL. 79 Supl. IV - 2019

80° Aniversario



MEDICINA

Volumen 79, Supl. IV, págs. 1-338

medicina

BUENOS AIRES, VOL. 79 Supl. IV - 2019

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

MEDICINA (Buenos Aires) – Revista bimestral – ISSN 0025-7680 (Impresa) – ISSN 1669-9106 (En línea)

REVISTA BIMESTRAL
Registro de la Propiedad Intelectual N° 02683675
Personería Jurídica N° C-7497
Publicación de la Fundación Revista Medicina (Buenos Aires)
Propietario de la publicación: Fundación Revista Medicina
Queda hecho el depósito que establece la Ley 11723

Publicada con el apoyo del Ministerio de Ciencia, Tecnología e Innovación Productiva.
MEDICINA no tiene propósitos comerciales. El objeto de su creación ha sido propender al adelanto de la medicina argentina. Los beneficios que pudieran obtenerse serán aplicados exclusivamente a este fin.
Aparece en *MEDLINE (PubMed), ISI-THOMSON REUTERS (Journal Citation Report, Current Contents, Biological Abstracts, Biosis, Life Sciences), CABI (Global Health), ELSEVIER (Scopus, Embase, Excerpta Medica), SciELO, LATINDEX, BVS* (Biblioteca Virtual en Salud), *DOAJ, Google Scholar y Google Books.*
Incluida en el Núcleo Básico de Revistas Científicas Argentinas del CONICET.

Directores Responsables:
Basilio A. Kotsias, Eduardo L. De Vito, Isabel Narvaiz Kantor, Guillermo B. Semeniuk

Secretaría de Redacción: Ethel Di Vita, Instituto de Investigaciones Médicas Alfredo Lanari, Combatientes de Malvinas 3150, 1427 Buenos Aires, Argentina
Tel. 5287-3827 Int. 73919 y 4523-6619
e-mail: revmedbuenosaires@gmail.com – http//: www.medicinabuenosaires.com

Vol. 79, Supl. IV, Noviembre 2019

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

LXIV Annual Meeting of
Sociedad Argentina de Investigación Clínica (SAIC)

LI Annual Meeting of
Asociación Argentina de Farmacología Experimental (SAFE)

XXI Annual Meeting of
Sociedad Argentina de Biología (SAB)

XXXI Annual Meeting of
Sociedad Argentina de Protozoología (SAP)

IX Annual Meeting of
Asociación Argentina de Nanomedicinas
(NANOMED-ar)

VI Regional Scientific Meeting of Asociación Argentina de Ciencia y Tecnología de Animales de Laboratorio (AACyTAL)

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

LA TAPA

Antonella Ricagni. **Atardecer en la calle**

Técnica: Aguatinta /aguafuerte. Año 2011. Medidas: 21 x 29 cm. Gentileza del autor.

Antonella Ricagni es Licenciada en Artes Visuales, con orientación en Grabado. Ha ejercido la docencia en Artes Plásticas en el nivel primario. Trabajó en varios museos como orientadora de sala y tallerista. Es escenógrafa egresada de la Escuela Metropolitana de Arte Dramático (EMAD). Ha realizado una residencia artística en México especializada en Xilografía.

Actualmente es docente en la materia Ilustración, en la carrera de Diseño Gráfico en la Facultad de Arquitectura, Diseño y Urbanismo, Universidad de Buenos Aires, y en Plástica y Tecnología en varias instituciones educativas en la ciudad de Buenos Aires.

Fuentes: https://www.behance.net/antoricagn37bb
https://www.linkedin.com/in/antonella-ricagni-4b48a0120/

COMISIONES DIRECTIVAS 2019

SAIC

Presidente
Dra. Mónica Costas

Vicepresidente
Dra. Cristina Carrillo

Secretaria
Dra. Gabriela Marino

Tesorero
Dr. Pablo Azurmendi

Prosecretaria
Dra. María Laura Ruiz

Vocales
Nodo FCEN
Dra. Geraldine Gueron
Nodo FFyB
Dra. Mariel Nuñez
Nodo Facultad de Medicina
Dr. Guillermo Keller
Nodo NCO
Dr. Carlos Laino
Nodo Región Sur
Dr. Ezequiel Lacunza
Nodo IByME-INGEBI-UCA
Dra. Flavia Saravia
Nodo INFICA
Dr. Marcelo Choi
Nodo Hospital de Clínicas
Dra. Florencia Giliberto
Nodo CEDIE
Dra. Mariana Tellechea
Nodo Hospital Garrahan
Dra. María Foncuberta
Nodo Academia Nacional de Medicina
Dra. Stella Ranuncolo
Nodo CEFYBO
Dr. Fernando Correa
Nodo Roffo
Dra. Mariana Callero

Revisores de Cuentas
Dra. Graciela Cremaschi
Dra. Andrea Randi

Secretaria Administrativa
Ivana Rossetto

SAFE

Presidente
Dr. Ana Genaro

Vicepresidente
Dr. Carlos Reyes Toso

Secretaria
Dra. Gabriela Acosta

Tesorera
Dra. Miriam Wald

Vocales
Dr. Santiago Daniel Palma
Dr. Ventura Simonovich
Dra. Lucía Fuentes

Revisores de cuentas titulares
Dra. Graciela Balerio
Dra. Wanda Novak

Revisores de cuentas suplentes
Dra. Patricia Bonazzola
Dra. Maria Palumbo

Secretaria Administrativa
Sra. Susana Gatti Maunas

NANOMED-ar

Presidente
Dra. Hebe Durán

Vicepresidente
Dra. Romina Glisoni

Secretaria
Dra. Leticia Higa

Tesorera
Dra. Julia Altube

Vocales titulares
Dr. Eder Romero
Dra. Mariela Agotegaray

Vocal suplente
Dra. Priscila Schilrreff

Revisora de cuentas titular
Dra. Marisa Taverna Porro

Revisora de cuentas suplente
María José Morilla

SAB

Presidente
Dra. Fernanda Parborell

Vicepresidente
Dra. Débora Cohen

Secretaria
Dra. Griselda Irusta

Tesorera
Dra. Isabel Lacau

Vocales titulares
Dra. Silvina Pérez Martínez
Dra. Mónica Muñoz de Toro
Dra. Clara Marín Briggiler

Vocales suplentes
Dra. Leandro Miranda
Dr. Pablo Cética

AACYTAL

Presidente
Ernesto Gulin

Vice-Presidente
Eliana Cicale

Secretario
Gabriel Pinto

Pro-secretaria
Marina Snitcofsky

Tesorera
Graciela Lammel

Pro-Tesorero
Gustavo Chapo

Vocales Titulares
Marcelo Asprea
Federico Alloatti
Marianela Lewicki
Angelica Miranda
Adela Rosenkranz
Eduardo Caturini

Vocales suplentes
Hugo Ortega
María Ines Zerba

Revisores de Cuentas
Mónica Lamer
Mariana Ríos

SAP

Presidente
Dra. Adelina Riarte

Vicepresidente
Dra. Fernanda Frank

Secretaria
Dr. Mónica Esteva

Pro-secretaria
Dra. María Belaunzarán

Tesorera
Dra. Silvia Longhi

Pro-Tesorera
Dra. Carolina Carrillo

Vocales
Dra. Karina Gómez
Dra. Catalina Dirney Alba Soto
Dra. Silvina Wilkowsky
Dra. Vilma Duschak

Comité científico
Presidente
Guillermo D. Alonso

Vice-Presidente
Vanina Alvarez

Miembros
Javier de Gaudenzi
Alan Talevi
Karina Gomez
Marisa Fernandez
Carolina Poncini
Natalia de Miguel
Alejandro Schijman
María Victoria Cardinal

HCS

Representante
Alejandro Adams

completely prevented the fructose-induced decrease in all of these parameters. Additionally, possible changes in the expression of the two major transcription factors involved in osteogenic and adipogenic differentiation, Runx2 and PPARgamma, as well as characteristic differentiation markers such osteocalcin and dentinsialoprotein, were evaluated by RT-PCR. It was found that DPPC-F presented a lower Runx2/PPARgamma ratio than de DLPC-C, whereas DPPC-CM and DPPC-FM presented similar values of ratios than control. At last, the expression of osteo/odontogenic markers decreased in DPPC obtained from group F, while treatment with metformin partially prevents these effects. These results confirm that the fructose-induced MS in rats decreases the proliferative and osteo/odontogenic potential of dental pulp progenitor cells. They also show that these deleterious effects can be completely or partially prevented by prolonged treatment with oral metformin.

0282 - MITOCHONDRIAL PHOSPHOENOLPYRUVATE CARBOXYKINASE IS EXPRESSED IN PP- BUT NOT BETA-CELLS FROM HUMAN PANCREATIC ISLETS

Ahilyn ESCOBAR(1) | Fernando MARTÍNEZ(1) | Francisco WESTERMEIER(2) | Rodrigo GATICA(3) | Francisco NUALART(1) | **Romina Paola BERTINAT** (1)

UNIVERSIDAD DE CONCEPCION (1); FH JOANNEUM GESELLSCHAFT MBH UNIVERSITY OF APPLIED SCIENCES, INSTITUTE OF BIOMEDICAL SCIENCE (2); ESCUELA DE VETERINARIA, FACULTAD DE CIENCIAS, UNIVERSIDAD MAYOR (3)

Abstract/Resumen: Mitochondrial phosphoenolpyruvate carboxykinase (M-PEPCK) is a cataplerotic enzyme that positively modulates glucose-stimulated insulin secretion in rodent beta-cells. However, M-PEPCK expression has not been addressed in human beta-cells. We hypothesized that M-PEPCK is expressed in human beta-cells, and used immunohistochemical techniques to prove it, comparing with rodent pancreas. For this end, pieces of pancreas from Sprague Dawley male rats, C57BL/6 beta-cell-ablated male mice, and cancer patients were fixed in formalin. Multiple immunofluorescence analysis of M-PEPCK and hormones specifically secreted by each cell population in the Langerhans' islets revealed the expression of M-PEPCK in insulin-producing beta-cells in both rat and mouse pancreas, as previously reported. However, we detected higher levels of M-PEPCK immunoreactivity in another endocrine cell population: PP-cells that secrete pancreatic polypeptide (PP). In fact, the same analysis in beta-cell-ablated mice (due to exclusive expression of human receptor for diphtheria toxin in beta-cells, and death of beta-cells upon treatment of mice with diphtheria toxin) showed that M-PEPCK reactivity and co-localization with PP is still high in the pancreas lacking beta-cells. Most significantly, we observed that M-PEPCK co-localizes with PP but not with insulin in biopsies from cancer patients. We conclude that M-PEPCK is not expressed in beta-cells from human pancreas, suggesting that extrapolation of data about M-PEPCK function in rodent pancreas should be reconsidered. In contrast, higher levels of M-PEPCK in PP-cells from both human and rodent pancreas suggest that this enzyme plays a role in PP secretion. This is the first study where the expression of M-PEPCK is addressed in human pancreas. Further studies are needed to understand M-PEPCK contribution to PP-cell metabolism. We postulate that M-PEPCK modulates PP production and/or secretion, contributing to nutrient homeostasis. This hypothesis is currently under investigation.

0288 - THE HYPOTHALAMIC EXPRESSION OF GONADOTROPIN-RELEASING HORMONE IS MODULATED BY THE COMBINED ACTION OF GLUTAMATE AND ESTRADIOL IN THE SOUTH AMERICAN PLAINS VIZCACHA (LAGOSTOMUS MAXIMUS).

Victoria FIDEL | Pablo Ignacio Felipe INSERRA | Sofia PROIETTO | Santiago Andrés CORTASA | Alejandro Raúl SCHMIDT | María Clara CORSO | Alfredo VITULLO | Julia HALPERIN | Verónica Berta DORFMAN

CENTRO DE ESTUDIOS BIOMÉDICOS, BIOTECNOLÓGICOS, AMBIENTALES Y DE DIAGNÓSTICO-UNIVERSIDAD MAIMÓNIDES

Abstract/Resumen: Lagostomus maximus shows peculiar reproductive features like reactivation of the reproductive axis during pregnancy with follicular recruitment and ovulation. Previously, we showed that hypothalamic expression of gonadotropin-releasing hormone (GnRH) of vizcacha correlates with serum estradiol (E2), estrogen receptors (ER) and N-methyl-D-aspartic acid receptor (NMDAR) expression during gestation. In addition, we observed that glutamate (GLU) down regulates GnRH delivery through NMDAR. Here we investigated the interaction between E2 and NMDA over GnRH expression in the hypothalamus of vizcachas in order to analyze their role on GnRH expression during gestation. We developed two approaches: 1- Hypothalamic explants of non-pregnant adult female vizcachas incubated with: a) GLU with or without (±) GLU receptors antagonists, b)NMDA ± NMDA-R antagonist CGP, c) E2 ± ERa or ERb antagonists, d) E2 ± NMDA ± CGP. GnRH mRNA levels were studied by RT-PCR; n=5/group. 2- Non-pregnant adult vizcachas ovariectomized (OVX) and treated with E2 (5 ug/kg); n=5/group. GnRH and NMDAR1 hypothalamic expression was evaluated by immunohistochemistry. We determined significant induction of GnRH mRNA expression by E2 and REa agonist related to control and REb agonist ($p<0.01$). In the contrary, we observed a significant decrease in GnRH mRNA levels induced by NMDA, and it was canceled by CGP ($p<0.005$). The combination of E2 with NMDA did not induce significant changes. On the other hand, hypothalamic protein expression of GnRH and NMDA showed significant increments of both neuromodulators in arcuate nucleus and medial eminence of OVX+E2 related to OVX and SHAM ($p<0.05$). These results suggest that GnRH expression is modulated by the combined action of E2 and GLU with opposite effects. This could represent a fine system of regulation of hypothalamic function over the reproductive axis of the female vizcachas.
Supported by Fundación Científica Felipe Fiorellino, CONICET-PIP110/14 and MINCyT-PICT1281/2014 grants.

0297 - SIMILAR EFFECTS OF ESTRONE AT VASCULAR AND BONE TISSUES: ¿RISK OR BENEFIT?

María Carla CRESCITELLI | María Belén RAUSCHEMBERGER | Virginia MASSHEIMER

INBIOSUR, DEPARTAMENTO DE BIOLOGÍA, BIOQUÍMICA Y FARMACIA, UNS-CONICET

Abstract/Resumen: It is known that the decrease in estrogen levels during menopause negatively affects bone tissue and vascular function. We have previously shown that E_1 modulates the cellular events involved in atherogenesis such as vascular smooth muscle cells (VSMC) proliferation and migration, and monocyte/platelet adhesion to endothelium. Vascular calcification represents the advanced stage of atherogenesis, where VSMC transdifferentiation on bone like cells plays a crucial role. The aim of this work was to compare the effect of 10 nM E_1 (48 h) on murine calvarial osteoblasts (OB), and on vascular smooth muscular cells transdifferentiated into osteoblasts (VSMC-OB) by incubation in a procalcification medium (10 mM glycerophosphate) for 21 days. We focused our attention on cell differentiation. E_1 increased cell proliferation on both cellular types, either using MTT assay (39 vs. 27% a/each C, OB vs. VSMC-OB, $p<0.05$) or cell counting technique (41 vs. 28 % a/each C, $p<0.05$; OB vs. VSMC-OB). On OB, E_1 treatment stimulated ALP activity (4.64 ± 0.32 vs. 3.37 ± 0.25; E_1 vs C; $x10^{-2}$ IU/mg prot., $p<0.001$), calcium deposition (40.2 % a/C, $p<0.05$; alizarin red staining) and collagen in extracellular matrix visualized by Sirius red staining (21 % a/C, $p<0.05$). Similar results were obtained when CMLV-OB cells were employed. E_1 enhanced ALP activity (3.72 ± 0.25 vs. 3.00 ± 0.14; E_1 vs. C,

$x10^{-2}$ IU/mg prot, p<0.001) and the number and size of calcification nodules in extracellular matrix (56 % a/C, p<0.05). Simultaneously, E_1 decreased calcium content in culture medium (735.0 ± 30.5 vs. 468.2 ± 21.1; C vs. E_1, µg Ca/mg prot., p<0.001). Indeed E_1 enhanced extracellular collagen deposition in CMLV-OB cells (19 % a/C, p<0.05). The results presented in this work show a similar action of E_1 in both cellular systems. The data suggest an opposite physiological relevance: a beneficial action at bone level favouring osteoblastogenesis and, a deleterious one at vascular homeostasis, promoting vascular calcification.

0308 - ESTROGEN RECEPTOR AND ENDOTHELIAL DYSFUNCTION

María Ivone VALLE (1) | María Belén RAUSCHEMBERGER(1) | W ESPECHE(2) | M SALAZAR(2) | R PLUNKETT(3) | Virginia Laura MASSHEIMER(1)

INBIOSUR, DEPARTAMENTO DE BIOLOGÍA, BIOQUÍMICA Y FARMACIA, UNS-CONICET (1); UNIDAD DE ENFERMEDADES CARDIOMETABÓLICAS. HOSPITAL SAN MARTÍN, LA PLATA (2); SERVICIO DE ENDOCRINOLOGÍA. HOSPITAL PRIVADO DR. RAÚL MATERA. BAHÍA BLANCA (3)

Abstract/Resumen: Estrogen receptor (ER) plays key role on vascular homeostasis. Alterations of ER signaling could conduct to vascular dysfunction, impairment of angiogenesis and platelet aggregation and activation. Preeclampsia (PE) is a pregnancy disease that exhibits these features. We have previously reported that the ER activation by estrone (E1), second natural estrogen, stimulates vasodilators synthesis (NO and PGI2) and inhibits platelet aggregation. This work presents basic (1) and clinical (2) results about the association of ER and vascular dysfunction. 1) Endothelial cells (EC) were incubated with E1 10 nM for 24 h. (2) We tested the existence of association between polymorphisms of ESR1 with PE in a high-risk pregnant women population. Polymorphic variants were studied by RFLP-PCR, employing PvuII and XbaI and resolved by electrophoresis in agarose gels. The in vitro treatment of EC with E1 shows that the hormone stimulated EC growth (91 % a/C, p<0.001) and VEGF synthesis. ICI 182780 (RE antagonist), suppressed E1 action. In pregnancy women, the frequencies determined by PvuII was 29 % 1 (C/T); 59 % 2 (T/T) and 12 % 3 (C/C). When XbaI was used the distribution was 35.3 % A (A/G); 2 % B (A/A) and 62.7 % C (G/G). The main haplotype determined was 2C. In contrast, in young women without PE risk the main was 1A. PE pregnant women exhibits a significant reduction in plasmatic NO levels respect to the whole pregnant population (0.15 ± 0.024 vs. 0.29 ± 0.040 mM NO/plasma resp.) Results shows that E1 and ER have relevant action on cellular processes involved in endothelial dysfunction. Since the distribution of ER genotypes is different among young women with and without risk of endothelial dysfunction, it could suggest a possible clinical utility of these markers as PE risk predictors.

0403 - ONCOGENIC POTENTIAL OF PROLONGED GH-ADMINISTRATION IN ADULT MICE LIVER

Nadia Sofía CICCONI | Magalí Cecilia CERCATO | Julieta Rocio CEBRON | Mariana Andrea BOJORGE | Lorena GONZALEZ | Johanna Gabriela MIQUET | Ana Isabel SOTELO

INSTITUTO DE QUÍMICA Y FISICOQUÍMICA BIOLOGICA (IQUIFIB) UBA-CONICET

Abstract/Resumen: Growth hormone (GH) is given to children with growth impairment and to adults under catabolic states, even if they are not GH-deficient. Chronic exposure to elevated GH levels induces pro-oncogenic liver pathology; GH-overexpressing transgenic mice develop liver tumors at advanced ages. We had evaluated the effect of 5-week GH-treatment, given with the hepatic tumor inductor diethylnitrosamine (DEN), on tumor formation in growing male mice liver. GH did not promote tumor formation, nor did GH given with DEN increase the number of hepatic lesions in growing mice. The aim of this study was to assess if the same prolonged GH pharmacological treatment would induce any alterations when given to 5 month-old mice. Livers (n= 7-9) were collected at 48 weeks of age and visually inspected. GH-treatment alone did not induce visible lesions. DEN treatment induced liver tumor formation, whereas combination with GH did not promote further tumor development. Microscopical evaluation revealed that only DEN-treated groups exhibited dysplastic foci, although non-significant differences were attained with GH-treatment. The number of hepatocytes per microscopic field was increased in the dysplastic foci compared to the surrounding tissue (p<0.05), denoting smaller cell size. Mice treated with GH exhibited a significantly lower hepatocyte count per microscopic field (p<0.05), 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

Abstract/Resumen: 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