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La Tapa (Ver p 5)

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LA TAPA

María de Luján Alvarez. Ludueña

Técnica: óleo sobre tela

Medidas: 60 x 40 cm, año 2016

Gentileza de la autora

La obra de tapa refleja un lugar típico rosarino. El arroyo Ludueña nace en los campos de las afueras de Rosario y finaliza en el barrio Arroyito de la ciudad, donde desemboca en el Río Paraná.

María de Luján Alvarez es Bioquímica y Doctora en Ciencias Biológicas. Es investigadora adjunta (CIC-CONICET) en el Instituto de Fisiología Experimental (IFISE-CONICET) y docente en el área Morfología de la Facultad de Ciencias Bioquímicas y Farmacéuticas de la Universidad Nacional de Rosario (UNR). Alumna del taller de arte Tunkeyén, estudió con la pintora rosarina Ana Petrini. Ganó el segundo premio en el 12º Salón de Pintores Nóveles de la Sociedad Argentina de Artistas Plásticos de Rosario (2004), el primer premio en el 2º Salón Pintando Argentina de Rosario (2010), una mención al trabajo realizado en el 2º Encuentro de Pintores de Rosario organizado por la Asociación Cultural Museo Ambrosio Gatti (2018) y el tercer premio en el Concurso de Pinturas 150 años de la Sociedad Filantrópica Suiza (2018). Participa frecuentemente en muestras colectivas de diferentes salones pictóricos rosarinos y sus obras han sido expuestas en espacios de arte organizados por CONICET y la UNR.

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tween cell clusters of the scRNA-seq data from Bach et. al., 2017. Preliminary results from our analysis show a significant increase in the expression of the lipogenic *Srebf1, Thrsp, Fasn* and *Me1* genes in mammary epithelial luminal progenitors in the transition from of virgin mice to lactation, and a concomitant decrease of the same genes from lactation to post-involution. These results support the role of the LXR as an important modulator of the lipid homeostasis in the lactating gland, modulating milk requirements to feed the newborn.

CARDIOVASCULAR Y RESPIRATORIO

26. (18) **TRIIODOTHYRONINE (T3) IMPROVES POST-ISCHEMIC MECHANICAL RECOVERY AND MITOCHONDRIAL FUNCTION PRESERVATION BY ENHANCING AMP-ACTIVATED PROTEIN KINASE (AMPK) ACTIVATION.**

Romina Hermann[1,2], María de las Mercedes Fernández Pazos[1], Mailen Florencia Córdoba[1], Federico Joaquín Reznik[1], Victoria Evangelina Mestre Cordero[1,2], Débora Elisabet Vélez[1,2], Andrea Fellet[1,2], María Gabriela Marina Prendes[1,2]

1-Universidad de Buenos Aires. Facultad de Farmacia y Bioquímica. Departamento de Ciencias Biológicas. Cátedra de Fisiología. Buenos Aires, Argentina.

2-CONICET - Universidad de Buenos Aires, Instituto de Química y Metabolismo del Fármaco (IQUIMEFA). Buenos Aires, Argentina.

Experimental evidence has shown that T3 can regulate cardioprotective signaling pathways, increasing myocardium resistance to ischemia-reperfusion injury. Although novel studies have suggested that T3 enhances the activation of AMPK, a key enzyme regulator of energy balance, its role has not been elucidated yet.

In the present study the role of AMPK in the effects exerted by acute treatment with T3 on ischemic-reperfused myocardium was investigated. For this aim, rat left atria were subjected to 75 min simulated ischemia (I)-75 min reperfusion (R), in the presence of T3 (60 nM) and AMPK inhibitor, Compound C (CC; 10 μM). ANOVA, followed by Tukey, n=8/group.

Results showed that CC prevented the increase of AMPK activation induced by T3 (End stabilization period (ESP):1.33±0.02, I-R:2.20±0.11*, I-R+T3:2.79±0.10*#, I-R+T3+CC:1.17±0.10 AU;*p<0.05 vs ESP, I-R+T3+CC; #p<0,05 vs I-R). At the end of R, CC abolished the increase of contractile function recovery (Peak force (%) I-R:36±3, I-R+T3:51±2*, I-R+T3+CC:39±4;*p<0.05) and cellular viability conservation produced by T3 treatment (I-R:66±4, I-R+T3:79±3*, Is-Rs+T3+CC:54±3 %;*p<0.05). CC also prevented mitochondrial ultrastructure preservation exerted by T3, as well as mitochondrial ATP production rate and tissue ATP enhancement (I-R:24±1, Is-Rs+T3:59±6*, I-R+T3+CC:40±2 nmol/min/mg mitochondrial protein; I-R:420±52, I-R+T3:608±94*, I-R+T3+CC:266±46 pmol/mg protein;*p<0.05). In addition, CC reverted the increase induced by T3 in the calcium amount required to trigger massive mitochondrial calcium release (I-R:77±9, I-R+T3:114±12*, I-R+T3+CC:82±8 nmol/mg protein;*p<0.05) and the phosphorylation and inactivation of GSK-3β, master switch enzyme that limits mPTP opening (I-R:1.5±0.2, I-R+T3:2.2±0.1*, I-R+T3+CC:1.6±0.2 AU;*p<0.05).

Results suggest that AMPK is involved, at least in part, in the protective effects exerted by T3 in the ischemic-reperfused myocardium, contributing to mitochondrial structure and function preservation.

27. (251) **DIFFERENTIAL BINDING TO EXTRACELLULAR MATRIX COMPONENTS OF A NATURAL APOLIPOPROTEINA-I VARIANT ASSOCIATED WITH CARDIAC AMYLOIDOSIS**

Rosu S[1], Gisonno R[1], Cortez F[1], Calabrese G[2], Urbano B[3], Sanchez Donoso S[3], Ramella N[1], Tricerri A[1]

1 Instituto de Investigaciones Bioquímicas de La Plata (CONICET-UNLP), La Plata, Argentina

2 Facultad de Farmacia y Bioquímica, Cátedra de Biología Celular y Molecular, Universidad de Buenos Aires. CABA, Argentina

3 Departamento de Polímeros. Facultad de Ciencias Químicas. Universidad de Concepción, Concepción, Chile

Specific interactions of apolipoproteins with components of the extracellular matrix (ECM), as proteoglycans (PGs), are associated with amyloidosis or atherosclerosis. These interactions seem to depend on age, cellular differentiation, and pathological conditions, which might modify glycosaminoglycans (GAGs) composition.

We previously showed that apoA-I Arg173Pro (a natural mutant involved in cardiac amyloidosis) but not the wild type protein (Wt) bound heparin at pH 7.4 This indicates that selective interactions of this variant may occur with GAGs. To study the specific role of the matrix?s charge on the interaction of apoA-I with GAGs, we synthesized polymers having different ratios of sulfated (sodium 4-styrene sulfonate, (SSNa) or hydroxilated monomers (2-hydroxyethyl methacrylate, (HEMA)) and studied the binding of fluorescently labelled apoA-I Wt or Arg173Pro. We show that both proteins are highly retained as long as the negative charge increases (50% with $p \leq 0.05$ as negative charge of the SSNa increased from 0.25 to 0.75 M). In addition, Arg173Pro remained in the matrix 10 % more than Wt ($p < 0.01$), indicating that the retention of specific proteins in the ECM could be part of the pathogenicity. To analyze the differential interaction with GAGs, Wt or Arg173Pro were incubated in the presence of Dermatan Sulfate (DS) or sodium heparin (HEP). The samples (n=4), were centrifuged, and pellets and supernatants analyzed by polyacrylamide gel electrophoresis. GAGs were visualized by staining with toluidine blue and the proteins with Silver stain. We observed that Arg173Pro had more interaction with DS and HEP than the Wt variant, which is interesting, since both GAGs are associated with amyloid deposits and heart disease, respectively. We conclude that the interactions of apoA-I variants with GAGs offer a challenging field to understand, not only the pathology but also possible therapeutic strategies to treat this disease.

28. (252) **HUMAN APOLIPOPROTEIN A-I IN ATHEROSCLEROSIS. THE ROLE OF OXIDATION OR NATURAL VARIANTS SYNERGIZING ITS DYSFUNCTION.**

Gisonno R[1,2], Díaz Ludovico I[1,2],Rosú S [1,2], Cortez F [1],Gonzalez M[1,2],Garda H[1,2], Gorgojo JP[3],Rodriguez E[3],Tricerri A[1,2],Ramella N[1,2]

1 Instituto de Investigaciones Bioquímicas de La Plata (INIBIOLP), Argentina

2 Facultad de Ciencias Médicas y Facultad de Ciencias Exactas, Universidad Nacional de La Plata. Calle 60 y 120, La Plata. CP 1900, Argentina

3 Centro de Investigación y Desarrollo en Fermentaciones Industriales (CINDEFI), La Plata, Argentina

Oxidation of human high density lipoprotein and its major protein apolipoprotein A-I (apoA-I) was proposed to cause their failure to protect against cardiovascular disease. However, multiple and complex events might contribute to the breakdown of this protein to fulfill its protective role. To set light on this topic, we took advantage of the study of a natural variant with a deletion of the lysine 107 (K107del) associated with atherosclerosis and amyloidosis. We oxidized the variants by controlled incubation with H2O2 and determined structural and biological parameters by biophysical and biological approaches.

Both variants oxidized under these conditions preserved or even induced an increase in the lipid clearance with respect to untreated proteins (20 % with $p \leq 0.05$), and decreased the yield of the physiological dimeric conformations. Following 30-day incubation at 37oC K107del but not Wt acquired a well-defined fibrillar conformation which is the main signature of the amyloid pathology. These conformations (but not freshly folded proteins) activated neutrophils into the formation of neutrophil extracellular traps (NETs) ($p<0.05$), which was drastically elicited in the case of the K107del ($p<0.001$).

To initiate the search of possible pathways involved in cellular activation, we tested a cellular model of macrophages. Fresh and oxidized Wt promoted the increase of p62 ($p \leq 0.005$), a protein described as anti-inflammatory. In the presence of ATRA (an inhibitor of Nrf2-Keap pathway), only Wt effect was blocked (by 40%). Thus, it may be suggested that the oxidation of apoA-I resulted in the loss of one of its key functions. Altogether, our data support that post

translational apoA-I modifications (probably chronic and progressive) raised a protein conformation with significant loss of function and increased aggregation tendency. Oxidation may help favoring this conformation. The results learnt here strength a close association between amyloidosis and atherosclerosis.

29. (261) **COBALT CHLORIDE PROTECS THE HEART AFTER A GLOBAL ISCHEMIC INSULT.**

Gutierrez C[1], Farcy N[1], Bonazzola P[1] y Castilla R[1]
[1]Universidad de Buenos Aires. CONICET. Instituto Alberto C. Taquini de Investigaciones en Medicina Traslacional (IATIMET)

Introduction: Ischemia-reperfusion (I/R) is one of the main cardiovascular risk factors and leads to heart contractile and energy dysfunction. I/R-induced damage is reduced by ischemic postconditioning. $CoCl_2$ has properties to function as a postconditioning agent, since it can trigger transcriptional changes that resemble the response to a hypoxic event under normoxic conditions.
Objectives: To evaluate $CoCl_2$ as a postconditioning therapeutic tool after a myocardial and arterial I/R.
Materials and Methods: Isolated adult Wistar rats hearts were arterially perfused at 37ºC by Langendorff method, paced at 3 Hz, exposed to 30 min ischemia followed by 45 min reperfusion (R) in the presence or absence of 0.23 mM $CoCl_2$ which was maintained or removed after 20 min of R.
Aortic contractility was evaluated in an isolated organ bath trough incubation with cumulative noradrenaline (NA) doses. After NA washing, 20 min of simulated arterial ischemia (SI) and R with or without $CoCl_2$ was performed, and NA response was re-evaluated.
Results: During R, the presence of $CoCl_2$ did not alter the cardiac resting pressure, nor the perfusion pressure, but increased the developed pressure ($p<0.05$) until 20 min which then descend reaching controls values. This decrease was prevented when $CoCl_2$ was eliminated at 20 min of R. $CoCl_2$ in R increased the contractile economy (P/Ht) and decreased the cardiac damaged area ($p<0.05$) and the incidence of arrhythmias ($p<0.001$).
Post-SI arterial contractility increased at the lowest NA dose but not at higher ones. $CoCl_2$ in post –SI R did not affect arterial force, but decreased NA sensitivity (EC 50: control: $10^{-7.5}$, $CoCl_2$: $10^{-6.5}$ M) and the maximum contractile response.
Conclusion: The use of $CoCl_2$ after an ischemic event attenuates the cardiac damage produced at least during the first 25 min of R and reduces the arterial adrenergic contractile response. These results support the use of $CoCl_2$ as a potential cardioprotective tool of clinical relevance.

30. (267) **H_2O_2, NO AND $ONOO^-$ IN THE CARDIAC MITOCHONDRIAL DYSFUNCTION IN A TYPE 1 DIABETES MODEL**

Rukavina-Mikusic IA[1,2], Rey M[1], Bombicino SS[3] and Valdez LB[1,2]
[1]Universidad de Buenos Aires, Facultad de Farmacia y Bioquímica, Cátedra de Fisicoquímica
[2]CONICET, Instituto de Bioquímica y Medicina Molecular (IBIMOL; UBA-CONICET), Fisicoquímica
[3]Universidad de Buenos Aires, Facultad de Farmacia y Bioquímica, Cátedra de Inmunología e Instituto de Estudios de la Inmunidad Humoral, Prof. Ricardo A. Margni (IDEHU, UBA-CONICET)

AIM: To study the changes of mitochondrial production rates and/or steady-state concentrations ($[X]_{ss}$) of $O_2^{\cdot-}$, H_2O_2, NO and $ONOO^-$ in the temporal evolution of cardiac mitochondrial dysfunction in a type 1 diabetes model. **METHODS:** Diabetes was induced by a single dose of Streptozotocin (STZ, 60 mg/kg, ip) in male rats. Glycemia (mg/dl) was determined after 72 h (C:130 ± 5; DM:415 ± 23). The animals were sacrificed after 10 or 28 days of STZ-injection (7 or 25 days of hyperglycemia). Mn-SOD activity, and H_2O_2 and NO production rates were determined in the cardiac mitochondrial fraction. $[O_2^{\cdot-}]_{ss}$,$[NO]_{ss}$ and $ONOO^-$ generation were estimated from experimental data. **RESULTS:** When animals were sacrificed 10 days after STZ-injection, heart mitochondrial NO (30%) and H_2O_2 (117%) productions were higher and Mn-SOD activity was lower (15%) than control values. Moreover, mitochondrial $[O_2^{\cdot-}]_{ss}$ was 2.5-fold higher in heart from diabetic rats, along with a 30% increase in $[NO]_{ss}$. Thus, $ONOO^-$ production rate resulted 3 times higher. When animals were subjected to 25 days of hyperglycemia, Mn-SOD activity was really reduced (50%). While H_2O_2 generation was extremely augmented (128%), the increase in NO generation (23%) was similar to the one observed at 7 days. Increases in $[O_2^{\cdot-}]_{ss}$ (350%), $[NO]_{ss}$ (25%), and $ONOO^-$ production (450%) were obtained. Moreover, nitration of tyrosine residues of mitochondrial proteins was observed in diabetic animals sacrificed at day 28. **CONCLUSIONS:** Heart mitochondrial production rates of H_2O_2, NO and $ONOO^-$ were higher in diabetic than in control animals, both after 7 and 25 days of hyperglycemia. No difference in the increase -over the control values- of $[NO]_{ss}$ was observed over time, while a much greater rise in $[O_2^{\cdot-}]_{ss}$ was detected after 25 days of sustained hyperglycemia respect to the enhancement obtained at 7 days, intensifying the difference in $ONOO^-$ generation. Therefore, $ONOO^-$ generation rate is mainly controlled by $[O_2^{\cdot-}]_{ss}$ rather than by $[NO]_{ss}$

31. (334) **HYPERBARIC OXYGENATION THERAPY IN THE TREATMENT OF COVID-19 (PRELIMINARY REPORT)**

Keller GA[1,2], Colaianni I[1], Bartolome J[1], Garcia ER[1], Rombola N[1], Cannellotto M[3], Di Girolamo G[2], Di Salvo HE[1].
1. Hospital General de Agudos Donación Francisco J. Santojanni
2. Universidad de Buenos Aires, Facultad de Medicina, Instituto Alberto C. Taquini de investigaciones en Medicina Traslacional (IATIMET).
3. Asociación Argentina de Medicina Hiperbárica e Investigación (AAMHEI)

Introduction: Hyperbaric oxygenation therapy (HBOT) has been shown to reduce the production and release of pro-inflammatory cytokines. Its use in patients with CoViD-19 and hypoxemia in China showed promising results although its use was poorly evaluated. Methods: A randomized, controlled study was started, comparing standard care (Control Group which includes non-hyperbaric oxygen supply) versus standard care plus HBOT (Test). The HBOT was carried out with Biobarica chambers of national development and medium pressure (1.45 atm) in sessions of 90 minutes per day for at least 5 days. Baseline daily oxygen saturation (SatO2) breathing ambient air (FiO2 = 0.21%) prior to HBOT was recorded. Results: 18 patients were included (ratio 1:1). The comparison between the groups did not show significant differences in terms of clinical status, general compromise, laboratory, age, comorbidities or history. The evolution of the HBOT group showed a rapid increase in SatO2 with a significant difference between groups from day 4 onwards. On day 5, the HBOT group presented SatO2 94.4 ± 2.7 (91.0-98.0)% Vs 89,8±2,3 (87,0-94,0) in control group. The time to normalize oxygen Saturation was significantly shorter in the HBOT group [mean±SD (min-max)]: 3,3±1,4 (1,0-5,0) VS 5,8±1,4 (3,0-7,0) days (P=0,002). The ascending slope for SatO2 in HBOT group was significantly higher than the control group: 2,1±0,6 (1,3-3,2) VS 1,5±0,6 (0,8-2,8) %/day (P 0,04). No adverse reactions were recorded. Discussion: HBOT shown to be more effective than oxygen suppy at ambient pressure. Although the study continues to recruit individuals, initial clinical results show a clear beneficial effect.
Reference: Rui-Yong C et al. Efficacy analysis of hyperbaric oxygen therapy in the treatment of severe coronavirus disease 2019 patients. Acad. J. Second Mil. Med. Univ. ; 6(41): 604-611, 2020.

32. (429) **EFFECTS OF REMOTE ISCHEMIC PRECONDITIONING ON EARLY MYOCARDIAL POST-INFARCTION REMODELING**

Bin EP[1], Penas F[2], Goren N[2], Gelpi RJ[1], Donato M[1]
1. Institute of Cardiovascular Pathophysiology, Faculty of Medicine, University of Buenos Aires
2. Institute of biomedical research in retroviruses and AIDS

Introduction: It is known that remote ischemic preconditioning (rIPC) reduces infarct size in experimental models of myocardial infarction (MI); while its effect is controversial in the clinical setting. Particularly, the effect of rIPC on post-infarction ventricular remod-