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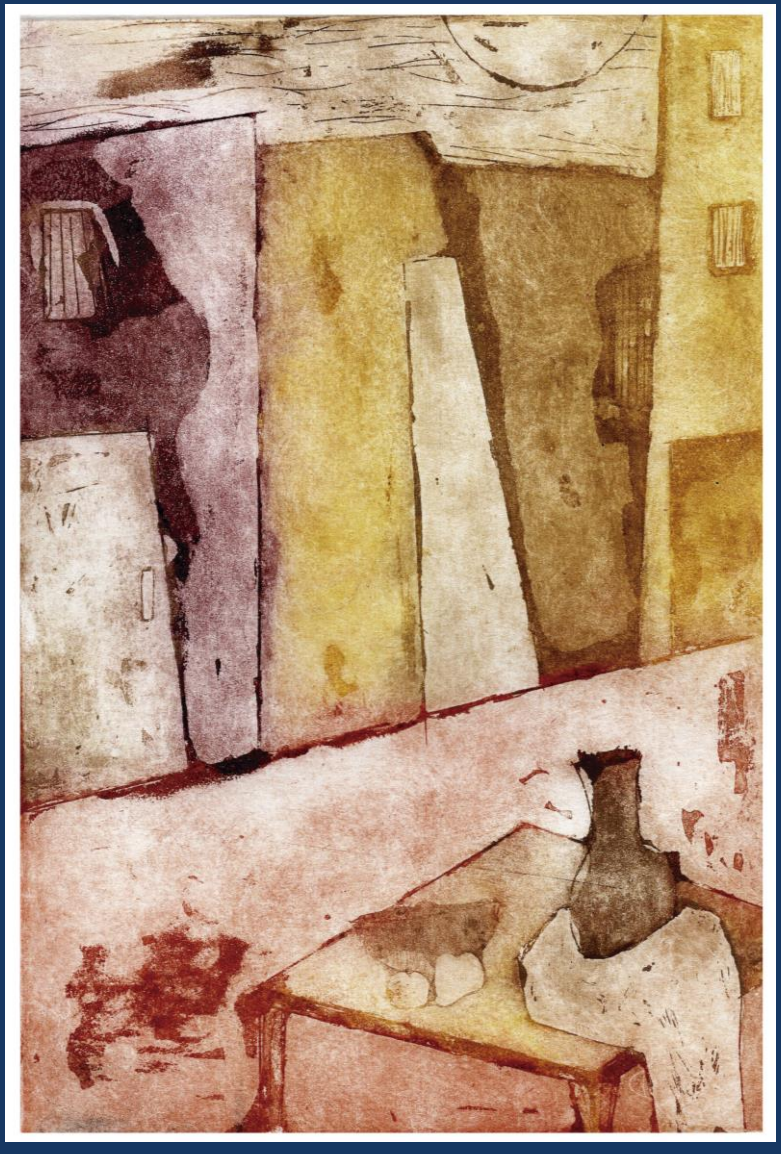

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La Tapa (Ver pág. 4)
Atardecer en la tarde
Antonella Ricagni

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11.7). In middle-aged groups, state 3 oxygen uptake was lower in Wt-I/R and Wt-PostC groups than Wt-Nx group (Wt-Nx: 176.0 ± 9.4 vs. Wt-I/R: 135.5 ± 9.6 and Wt-PostC: 113.9 ± 11.7). No differences appeared between Trx1 groups and the same was observed in DN-Trx1 groups. In conclusion, we found the cardioprotection mediated by PostC and Trx1 overexpression restore mitochondrial function in young mice but not in middle-aged mice.

0187 - CARDIOPROTECTIVE EFFECTS OF NEBIVOLOL ON ISCHEMIA/REPERFUSION IN HYPERTHYROID RATS: MECHANIC AND ENERGETIC STUDY

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CÁTEDRA DE FARMACOLOGÍA. FACULTAD DE CIENCIAS EXACTAS (1); CÁTEDRA DE FARMACOLOGÍA. FACULTAD DE CIENCIAS EXACTAS. UNLP / CONICET (2)

Abstract/Resumen: Nebivolol (Nbv) is a third-generation ß-blocker with high selectivity for ß1-adrenergic receptors and it induces vasodilation by stimulating endothelial nitric oxide synthase (NOS). These effects could be cardioprotective in hyperthyroidism. Now, we studied the mechanic and energetic effects of Nbv in euthyroid (EuT) and hyperthyroid (HypT) rat hearts exposed to severe ischemia-reperfusion (sI/R) without infarct. Rats were daily injected with 20 µg/kg triiodothyronine s.c. for 15 days. Isolated ventricles from either HypT or EuT rats were perfused in a calorimeter and exposed to sI/R (30 min I/ 45 min R). Left intraventricular pressure (P, mmHg) and total heat release (Ht, $mW.g^{-1}$) were measured. Perfusion with 0.03 µmol/l Nbv improved the postischemic contractile recovery (PICR) to 39.0 ± 4.4 % of pre-I of P (vs. 24 ± 3 % in HypT-C, *p<0.05) at 45 min R in HypT, and to 28 ± 3 (vs. 12 ± 5 % in EuT-C, *p<0.05) at 15 min R in EuT, without changing muscle economy (P/Ht). In both cases Nbv increased the diastolic contracture during R (LVEDP: 16 ± 4 and 20 ± 6 mmHg respectively, *p <0.05 vs. 0). To evaluate whether the nitric oxide (NO) participates in the cardioprotection of Nbv, the NOS were blocked by L-NAME before I. The effects of Nbv on PICR and P/Ht were significantly reversed by the pre-treatment with L-NAME without changing LVEDP in HypT and EuT. Other groups of rats were treated with 20 mg/kg Nbv daily administered in drinking water during 7 days. In EuT, Nbv significantly improved PICR to 78.8 ± 10.6% of pre-I (*p<0.05 vs. EuT-C) and P/Ht to 5.4 ± 0.9 $mmHg.g.mW^{-1}$ (*p<0.05 vs. 1.0 ± 0.4, EuT-C), without changing LVEDP. In HypoT, oral Nbv significantly improved PICR and P/Ht only at the end of R, but increased LVEDP during R. Results suggest that: a) Nbv was directly cardioprotective in HypT and EuT hearts exposed to sI/R and this effect was mediated by NO production from NOS; b) Hypothyroidism reduced the prevention of cardiac stunning induced by oral Nbv in EuT hearts.
This work was supported by UNLP-X-795 grant.

0204 - GENETIC DELETION OF GALECTIN 3 REDUCED SURVIVAL AND FAVORED MYOCARDIAL HYPERTROPHY IN AGED MICE

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Abstract/Resumen: Galectin 3 (Gal-3), a ß-galactosidase binding lectin widely expressed in the immune system, has proinflammatory and profibrotic effects. Gal-3 participates in ventricular remodeling (VR), hypertension, acute myocardial infarction and heart failure. However, the role of Gal-3 on mortality and cardiac remodeling associated with aging has not been previously studied. We aimed to study if genetic deletion of Gal-3 modifies survival rate and VR associated with aging. Wild type (WT, n= 54) and Gal-3 knockout (KO, n= 55) mice were in-housed and maintained on a 12 h light/dark cycle during 24 months. Animals had access to water and food ad libitum. Water and food consumption, body weight and survival rate were quantified. After 24 months of follow up, systolic blood pressure was measured by plethysmography. Heart (HW), lungs (LW), kidneys (KW) and spleen (SW) were weight and tibia length (TL) measured. Student's T tes t was used for comparison among groups and log rank test for survival curves (Kaplan meier). Results (Mean±SEM): Survival rate was significantly reduced in Gal-3KO mice as compared with WT (p= 0.02). Water and food consumption was higher in Gal-3 KO mice: 5.5 ± 0.1 ml/animal/week vs 3.3 ± 0.1 ml/animal/week (p<0.0001) and 2.7 ± 0.04 gr/animal/week vs. 2.2 ± 0.03 gr/animal/week (p<0.0001). Body weight and systolic blood pressure was similar between groups: 34.4 ± 0.5 vs. 35.3 ± 0.5 and 111.6 ± 2.9 vs. 114.3 ± 2.8 in WT and Gal-3KO respectively (p= NS). HW/TL and KW/TL were 8.0 ± 0.3 vs. 9.0 ± 0.3 (p<0.05) and 25.0 ± 0.9 vs. 34.0 ± 1.3 (p<0.05) in WT and Gal-3KO respectively. In summary, genetic deletion of Gal-3 during aging reduced survival associated with an increase in myocardial hypertrophy despite showing no effects in blood pressure. Further studies are neccesary to understand the mechanisms that links Gal-3 with myocardial hypertrophy and ventricular function during aging.

0218 - ASSOCIATION BETWEEN CLINICAL AND GENETIC DIAGNOSIS IN PATIENTS WITH LQT SYNDROME: IMPORTANCE OF GENETIC TESTING

Leonardo Raul DIONISIO (1) | Eugenio AZTIRIA(1) | Sofía STUPNIKI(1) | Leandro DYE(1) | Leonardo ONETTO(2) | Franco GREGORIETTI(2) | Roberto KEEGAN(2) | Guillermo SPITZMAUL(1)

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Abstract/Resumen: Long QT syndrome (LQTS) is a congenital genetic disorder that cause cardiac arrhythmia and sudden death. The genes more frequently implicated are those encoding for the K^+ channels KCNQ1 (40 - 45 %) and HERG (40 – 45 %), and the Na^+ channel Nav1.5 (5 – 8 %). Dysfunction in these channels leads to QT interval lengthening in ECG. Molecular identification of the causes of this disease contributes to better diagnosis, risk stratification and pharmacological treatment improvement. Our aim is to correlate clinical diagnosis with genetic variants of LQTS. We examined the LQT-associated genes KCNQ1, KCNH2 and SCN5A using gDNA extracted from 6 subjects. Five of them showed a prolonged QT interval on the ECG (>460 ms) while 1 first-degree relative presented a normal QT interval (<450 ms). The group (3 males and 3 females, 0-62 years old) included 1 with sudden cardiac death history under 40 years old and 1 with presyncope and documented polymorphic ventricular tachycardia. We amplified all exons from the 3 genes by PCR followed by sequencing. For KCNQ1 we found the uncommon variant c.1638G>A (p.Ser546=) in 2 subjects. In 1 patient we could not amplify exon 16, suggesting exon deletion. For KCNH2 we found the following variants: c.1692A>G (p.Leu564=) in 1 patient, c.1956T>C (p.Tyr652=) in 5 out of 6 cases and c.2690A>C (p.Lys897Thr) in 1 patient. Finally, we found the likely-pathogenic variant c.982C>T (p.Arg328Cys). For SCN5A no variants were detected at the tested exons. We found benign and pathological genetic variants in either KCNQ1 or KCNH2 genes of our population. No information about the exon 16 deletion for KCNQ1 as a pathological variant has been reported. To our knowledge, this is the first genetic test of LQTS performed in Argentina. Genetic characterization will impact on patient habits, avoiding risk situations such as sports, acute

stress or cardiotoxic drugs therapies. Moreover, these studies will enable to set patient-oriented pharmacological treatments.

0396 - TRYPANOSOMA CRUZI INFECTION INDUCES SLUG EXPRESSION IN HEART DURING CARDIAC REMODELING

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Abstract/Resumen: Chagas disease is caused by Trypanosoma cruzi infection. Chronic cardiac manifestation (CCC) is consequence of a cardiovascular remodeling (CR) process that elicit a dilated cardiomyopathy that can trigger heart failure. This process starts in heart and vessels during the acute phase of the infection, but the molecular mechanisms are poorly understood. We reported that inhibition of Wnt proteins reduces CCC severity n BALB/c mice. Despite smooth muscle cells (SMC) dedifferentiation and fibroblast (Fb) differentiation into myofibroblasts play an important role in CR, little is known about their contribution to this progression. Slug is a transcription factor crucial during development and pathogenesis. Our group demonstrated that Slug is associated with vascular remodeling and promotes SMC dedifferentiation. We also recently observed that in vitro TGF-ß; treatment of SMC and Fb promotes Slug downregulation. In this work, we tested the hypothesis that Slug is involved in CR process during T. cruzi infection. Consequently, we aimed to determine Slug expression in heart during acute and chronic T. cruzi infection in absence and presence of Wnt proteins secretion inhibition. We also evaluated the presence of myofibroblasts in heart. BALB/c mice were infected with 1,000 tripomastigotes and Slug was determined in hearts by q-PCR at different days post-infection (dpi). During acute phase of infection, a gradual upregulation of Slug that became statistically significant after 23 dpi respect to non-infected mice was observed (p= 0.0277). The expression of Slug remained upregulated in heart (p= 0.0330) during chronic phase of infection (180 dpi). Interestingly mice treated with a Wnt proteins secretion inhibitor (IWP-L6) increased TGF-ß; levels, partially blocked Slug upregulation and increased the number of myofibroblast in heart. Our results indicate that Slug could be involved in the regulation of CR during T. cruzi infection, probably by modulating SMC and Fb phenotype.

0471 - SUB-ANOREXIGENIC DOSES OF LEPTIN ACTIVATES THE LEPTIN-TRH CARDIAC HYPERTROPHIC PATHWAY.

Maia AISICOVICH | Mariano Luis SCHUMAN | Ludmila Soledad PERES DIAZ | Macarena ROSATI | Maria Silvina LANDA | Silvia Ines GARCIA

INSTITUTO DE INVESTIGACIONES MEDICAS A LANARI (UBA - CONICET)

Abstract/Resumen: Cardiac TRH (cTRH) induce left ventricular hypertrophy (LVH) and fibrosis. Additionally Leptine (Lep) induces TRH, until now only described in CNS. Our previous results on obese mouse models suggest that high Lep levels may impact cardiac tissue inducing cTRH expression and consequently stimulate LVH mediated by cTRH. So far this novel Lep-cTRH pathway has not been described in lean mice. We used C57 adult male mice to evaluate if sub-anorexigenic Lep doses could stimulate cTRH system hypothesizing that low doses of Lep could activate the Lep -cTRH pathway. For 3 weeks mice received subcutaneous leptin (10 ug/kg/day) or saline, with or without cTRH blockage by intracardiac injection of siRNA-TRH or siRNA-control (n= 7). As expected, Lep did not modify body weight, food intake or blood pressure during all the experiment, as there were no differences between those treated with Lep vs saline confirming the sub-anorexigenic Lep dose. As hypothesized we found that Lep significantly increased ($p<0.05$) cTRH mRNA expression (rtPCR) and precursor protein (WB). This increase was only observed in the group with the native TRH system compared to the group blocked by siRNA-TRH, confirming that the TRH blockade was successful. The Lep-induced TRH rise ($p<0.05$) brought the expression of hypertrophy and fibrosis markers (TGF-β, BNP and Collagens) as these results were only observed in the group with the active TRH system. We confirm on heart cells (H9C2, 3T3, primary myocytes) the direct induction of TRH (WB and RT-PCR) ($p<0.05$) by stimulating with different Lep doses (10 and 100 ng/ml) at different times. Finally we show for the first time that sub-anorexigenic Lep dose impact the heart and provoke an increase in hypertrophic and fibrosis markers mediated by cTRH. These results open the possibility that in obesity, cardiac alterations could begin prior to overweight, due to the initial slight increase in leptin levels that impact the heart inducing the cardiac TRH system.

0539 - THE LACK OF PROTECTION OF ISCHEMIC POSTCONDITIONING IN DYSLIPIDEMIC MICE IS RESTORED WITH N-ACETYLCYSTEINE TREATMENT

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Abstract/Resumen: Dyslipidemia per se exacerbated ischemia/reperfusion (I/R) injury, mainly caused by an increased of reactive oxygen species increases infarct size (MI). Ischemic postconditioning (IP) decreases MI, but we proved that in mice fed a high-fat diet (HFD, 12 weeks) with atherosclerosis in the early stages, the protective effect of IP is abolished. Therefore, the objective of our work is to evaluate the effect of treatment (T) with n-acetyl cysteine (NAC) restores the cardioprotective effect of IP in HFD mice. Male C57/BL6 mice (20 g) were divided into a control diet group (CD) and HFD group. In the last 3 weeks of feeding, a subgroup was treated with NAC (10 mM). Hearts were subjected to 30 min of I and 120 min R (Langendorff Technique). In the IP group, after I, 6 cycles of R/I were performed. The biochemical profile, histological alterations, ventricular function and MI were evaluated. Data are expressed as mean ± SEM and $p < 0.05$ was considered statistically significant (n= 6 per group). There was a slight decrease in weight in the NAC group without variation in daily caloric intake, cholesterol levels and triglycerides. NAC reduced the moderate hepatic steatosis in HFD group. As we expected, IP reduced MI in CD group (I/R: 54.3 ± 2.3 vs. IP: 36.5 ± 1.8; $p<0.05$), but this protection was abolished in the HFD-IP group (I/R: 66.4 ± 3.8 vs. IP: 62.1 ± 3.6). NAC T reduced MI in CD-IP+NAC with respect to the CD-I/R group and the CD-I/R+NAC group (I/R: 54.3 ± 2.3; I/R+NAC: 36.3 ± 4.6 vs. PI+NAC: 19.2 ± 3.3; $p<0.05$). On the other hand, in both NAC groups we observed a significant decrease in MI (HFD-I/R+NAC: 39.7 ± 4.5; HFD-IP+NAC: 26.4 ± 2.0; $p<0.05$ vs. HFD-IP and HFD-I/R). Regarding left ventricular function, we did not observe a significant difference between the groups after 30 min of I evaluated at 60 min after R. This study showed that alterations in the redox state in an early stage of atherosclerosis were enough to abolish the protective effect of IP, however T with NAC restored the cardioprotective effect of IP.