MSFE rose SBP vs CD in week 8 (p<0.0001). No differences were observed between SMFE and FE at all times. Basal ventricular function did not change between CD and treated groups. Contractile reserve was attenuated in MS and MSFE (p<0.05) vs CD. Ventricular relaxation rate was lower in MS, MSFE and MSAoV in response to isoproterenol. Conclusion: MS and FE load alone or in the presence of MS alter the cardiovascular system, inducing a rise in SBP and an impaired ventricular function, evidenced by contractile reserve and ventricular relaxation. AoV counteracts some of these effects, suggesting that proinflammatory condition as FE overload or SM induce cardiovascular damage that can be reverted by AoV.

47. (025) **ASSESSMENT OF CARDIOVASCULAR SAFETY IN MEDIUM PRESSURE HYPERBARIC OXYGENATION THERAPY**

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The "hyperbaric oxygenation therapy" (HBOT) is currently used as an adjunct therapy in many pathologies. Its therapeutic action is based on that hyperoxia at high pressure (2.4 atm) induces vasoconstriction and reduction of inflammation among others effects.
Recently, a more accessible Medium Pressure Hyperbaric Oxygenation therapy" (mHBOT) at 1.4 atm is also used as therapy that resulted equally effective at a lower cost.
However, up to date no studies have been carried out to support the cardiovascular safety of both treatments. For this reason, we studied the cardiovascular safety of mHBOT in rats subjected to a protocol equivalent to that applied in humans.
Male Sprague Dawley rats were submitted to 30 sessions of 60 min in a hypebaric chamber at 1.44 atm. and 100% O_2. Isolated hearts were perfused through aorta at 37ºC, paced at 3 Hz, and exposed to 30 min ischemia (I) followed by 45 min reperfusion (R). Simultaneous mechanical and heat measurements and the heart damaged area (trifeniltetrazolium) were evaluated.
Also aorta rings were superfused and the noradrenaline response was analyzed.
Hearts from mHBOT-treated rats showed an increase in resting pressure (RP) during ischemia (p<0.05) but no changes were observed in R.
An improvement (p<0.05) in post ischemic contractile recovery was observed in hearts from mHBOT-treated rats (65.4±12.9%) respect to control ones (33.5±6.1%) at 45 min R.
mHBOT did not alter total heat rate (Ht), but an increase (p<0.05) in contractile economy during R (120.9±20%) respect to control (53.2±8.9%) at 45 min was observed. Also mHBOT reduced the heart damage area induced by I/R.
Furthermore, arteries from mHBOT-treated rats showed similar response to noradrenaline than controls.
Conclusion: the mHBOT cardioprotects hearts from I/R injury acting as a preconditioning agent. The use of this therapy is safety for the cardiovascular system.

48. (067) **MITOCHONDRIAL DYNAMICS IN CARDIAC TISSUE DURING ACUTE ENDOTOXEMIA**

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Mitochondrial dynamics emerges as a compensatory mechanism in sepsis and endotoxemia, contributing to organ-cellular redox and energy management. In this work, we aimed to elucidate mitochondrial dynamics (fusion, fission, biogenesis and mitophagy) as a fundamental effector of cardiac tissue energy management in an experimental model of endotoxemia. Female Sprague-Dawley rats were subjected to low-grade endotoxemia (ip injection of LPS 0.5 mg kg -1 body weight) and severe endotoxemia (ip injection of LPS 8 mg kg -1 body weight) for 6 or 24 h. TEM analysis after 6 h low-grade endotoxemia showed higher number of mitochondria per cardiac tissue area and a shorter diameter. Also, structures compatible with damaged mitochondria were observed. In severe endotoxemia, highly damaged mitochondrial structures were observed, being these results associated with a 20% decrease in mitochondrial inner membrane potential, suggesting organelle dysfunction. Based on these results, expression of the main proteins involved in mitochondrial processes of fusion (OPA-1), fission (DRP-1), biogenesis (PGC-1α and mtTFA) and mitophagy (Pink-1 and Parkin), were studied. Low-grade endotoxemia exhibited a decrease in DRP-1 expression levels whereas Pink-1 expression was increased, both at 24 h treatment. Severe endotoxemia showed increases in OPA-1 and Pink-1 expression after 6 h treatment. The observed results suggest that the severity of endotoxemia relates to the degree of mitochondrial dysfunction and structural damage, and is linked to changes in mitochondrial dynamics as repairing processes. Our work presents novel results that contribute to elucidate the mechanisms by which endotoxemia, energy management, and mitochondrial architecture interact in cardiac tissue, arising this triade as a target to base future therapeutics for preserving this organ from inflammatory and oxidative damage.

49. (211) **EFFECT OF CARDIAC RENIN ANGIOTENSIN SYSTEM AXIS ON MALE AND FEMALE RATS EXPOSED TO MODERATE ZINC RESTRICCION DURING FETAL AND POSTNATAL LIFE.**

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Introduction: We have demonstrated that zinc deficiency during pregnancy and postnatal life induced cardiovascular alterations in males. Females did not show significant heart alterations.
Objective: Evaluate cardiac renin angiotensin system in adult rats exposed to zinc deficiency during growth.
Methods: Female Wistar rats fed either control (C30ppm) or low zinc (L8ppm) diets from pregnancy to offspring weaning. C male and female offspring continued with control diet (Ccm;Ccf respectively), whereas L male and female offspring fed control (Lcm;Lcf) or low zinc (Llm;Llh) diets. At day 81 we measured in left ventricle: Angiotensin (Ang) II, Ang(1-7) levels by radioimmunoassay, Angiotensin converting enzyme 1(ECA1),ECA2 and AT1 receptor (AT1R) expression by RT-qPCR; AT2R and AT1R expression by western blot and immunohistochemistry. Values are means±SEM, Two-way ANOVA, Bonferroni post-test.
*p<0.01vsCcm;§p<0.01vsLcm;‡p<0.01vsLlm; αp<0.05vsLcf;δp<0.05vsCcf,n=6.
Results: Llm and Lcm exhibited increased levels of AT1R mRNA and protein expression, and an increased in AngII(Ccm:4.4±1,2;Llm:12.0±1.9*§;
Lcm:2.6±0.2;Ccf:4.6±0.7;Llf:9.1±0.9δα; Lcf:6.5±2.1 nM/g).
Immunohistochemistry showed similar results in AT1R expression (Ccm:10.0±0.1;Llm:15.2±0.1*;Lcm:14.3±0.5*;Ccf:8.8±0.1;Llf:12.6±0.5δα;
Lcf:8.6±0.02 % of positive staining per area) and an increase of AT2R in female rats(Ccm:8.7±0.4;Llm:10.51±0.02;Lcm:11.2±0.3;Cch:15.9±0.2*;Llh:16.53±0.02‡;Lch:15.8±0.4§ % of positive staining per area). No changes were observed in Ang(1-7) content and ACE expression.
Conclusions: Zinc restriction during prenatal and postnatal growth exacerbated the cardiac Ang II-AT1R axis in adult male rats. An adequate zinc diet during postnatal life could not reverse these ef-