gen receptors in lung reveal that these hormones might play a key role in lung physiology. Obesity-mediated oxidative stress produced in adipose tissue is one of the main factors considered as oxidant source and inflammation mediator. The aim of this work was to study the effect of diet-induced obesity on the lung of androgen-deficient rats (castrated). Wistar male rats (200 ± 20 g) were separated in four groups: Control with normal diet (CoN), castrated with normal diet (KN), control with hypercaloric diet (CoOB) and castrated with hypercaloric diet (KOB) and sacrificed 30 days after castration. Biochemical parameters were analysed in serum and the expression of antioxidant enzymes and NOX-2, FOXO, HO-1 and RA in lung. ANOVA and Tukey test were used for statistical analyses. The results showed TBARS levels increased in KOB group compared to CoN ($p<0.001$) and KN ($p<0$, 01) groups, respectively. CAT activity was increased in KN ($p<0.05$) group. HDL levels increased in CoOB ($p<0.001$), KN ($p<0.001$) and KOB ($p<0.01$) groups. Both, urea and TG determinations were increased in KN ($p<0.001$); $p<0.01$ and KOB ($p<0.001$) groups. CL was increased in CoOB ($p<0.001$); KOB ($p<0.01$) compared to CoN group and increased in KN ($p<0.001$) and KOB ($p<0.001$) compared to CoOB group. CAT expression decreased in CoOB and KN groups, and RA expression increased in KN group compared to control group. Antioxidant enzymes NOX-2 and SOD-2 ($p<0.01$) and GPx-1($p<0,05$) increased in KOB group compared to control. FOXO-1 and HO-1increased in castrated group, with obesogenic diet ($p<0.01$). We previously demonstrated an important oxidative stress state in a castrated animal model. In conclusion, obesity added to androgen deficiency modifies different serum parameters. In fact, some inflammatory molecular pathways reveal a potential relationship between both situations (androgen deficiency-obesity).

345. (919) **ADIPOSE TISSUE AND INSULIN-RESISTANCE. BENEFICIAL EFFECTS OF GLP-1 AGONISTS**

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Insulin-resistance (IR) is characterized by adipose tissue (AT) expansion associated with extracellular matrix (ECM) remodeling. Metalloproteinases (MMPs) are endopeptidases involved in adipogenesis and angiogenesis, and they are proposed as pharmacological targets. Liraglutide (L), a glucagon-like peptide type 1 agonist, has emerged for the management of IR, and its effects on AT are still investigated. Aim: to evaluate the effect of L on MMPs activity in an animal model of IR. Methods: male Wistar rats (180-200 g) were divided in: Control (C, n=11) fed with standard diet, and sucrose rich diet group (SRD, n=14) fed with standard diet and sucrose 30% in drinking water during 15 weeks. Then, both groups were subdivided according to subcutaneous administration of L (0.6 mg/kg/day) for 5 weeks. The study was approved by the Ethic Committee-FFYB (UBA). Serum glucose, and lipid and lipoprotein profile were measured. Visceral AT (perirrenal, intestinal and epididymal) was removed and weighed. In epididymal AT (EAT) histological characteristics, MMPs activity by gelatinolytic zymography and antioxidants enzymes (SOD and Catalase) were evaluated. Results: as expected, SRD presented higher visceral AT mass ($p<0.05$), TG and glucose levels ($p<0.05$) than C. In SRD+L group, a significant decrease in body weight ($p<0.01$), EAT mass ($p<0.01$), TG ($p=0.045$) and glucose ($p=0.05$) levels compared to SRD was observed. As expected, SRD presented lower density of larger adipocytes than C ($p<0.05$). In turn, vascular density was lower in SRD ($p<0.05$ vs C). L decreased adipocyte size ($p<0.05$ vs SRD), as well as significantly increased vascular density in SRD+L ($p<0.001$ vs SRD). MMP-2 activity decreased in EAT from SRD ($p<0.05$ vs C) and increased in SDR+L ($p<0.05$ vs SRD). L decreased SOD activity in EAT in the SRD group ($p<0.01$) with no change in catalase activity. Conclusions: In IR, liraglutide would improve AT functionality by preventing disfavored features during MEC remodeling.

METABOLISM AND NUTRITION II

Friday, November 18, 9-10:30 hr
Chairs: Gustavo Hein - Gabriela Berg - Liliana Monasterolo

346. (147) **RENAL INFLAMMATION INDUCED BY HIGH FAT DIET IN RATS IS ASSOCIATED WITH RENAL DOPAMINERGIC SYSTEM ALTERATION AND SODIUM EXCRETION IMPAIRMENT**

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High fat diet (HFD) can cause metabolic alterations and a proinflammatory state that leads chronically to organ damage. In the kidneys, the renal dopaminergic system (RDS) plays an important role through its anti-inflammatory action by D2 like receptors (D2DR) activation. The aim of this study was to demonstrate the relationship between the RDS and natriuresis alterations with inflammation induced by a HFD in an animal model. For 8 weeks, Sprague Dawley rats (n=4-6/group) were randomized to HFD (50% w/w of bovine fat added to standard rodent diet) and control group (C) (standard rodent diet) and tap water to drink for both groups. The following determinations were performed; Insulinemia (commercial ELISA kit), L-dopa and dopamine urinary excretion (HPLC, L-dopa/dopamine index was calculated), expression of D2DR and pro-inflammatory molecules in renal cortex (Western blot), fibrosis (Picro Sirius red stain), and natriuresis through urinary sodium excretion (UNa.V). Results: L-dopa/dopamine index (HFD 3.7 ± 0.7 vs C 1.1 ± 0.1; $p<0.05$); insulinemia (HFD 3.2 ± 0.3 vs C 0.7 ± 0.1; $p<0.0005$); fibrosis % (HFD 28.0 ± 1.8 vs C 15.0 ± 0.8; $p<0.005$); NFkB (HFD 6.2 ± 0.8 vs C 2.2 ± 0.5; $p<0.05$); TGF-beta (HFD 8.3 ± 1.4 vs C 4.4 ± 0.6; $p<0.05$), were significantly improved in HFD vs C. D2DR (HFD 5.1 ± 0.8 vs C 12.3 ± 0.3; $p<0.005$) and UNa.V (mEq/24hs) (HFD 0.5 ± 0.1 vs C 1.8 ± 0.2; $p<0.0005$) were significantly decreased in HFD vs C. We observed positive correlations between L-dopa/dopamine index vs insulin ($r^2=0.90$, $p<0.001$), fibrosis % ($r^2=0.89$, $p<0.001$), NFkB ($r^2=0.83$, $p<0.001$); and fibrosis % vs insulin ($r^2=0.90$, $p<0.001$). Additionally, negative correlations between UNa.V vs L-dopa/dopamine index ($r^2=0.80$, $p<0.001$), D2DR ($r^2=0.93$, $p<0.001$), fibrosis % ($r^2=0.91$, $p<0.001$), and fibrosis % vs D2DR ($r^2=0.80$, $p<0.01$) were found. In conclusion, inflammation caused by a HFD leads to alterations of the RDS contributing to a reduction of renal sodium excretion.

347. (153) **OXDIDATIVE STRESS AND RESPIRATORY BURST STIMULATED BY URIDINE DIPHOSPHATE GLUCOSE (UDP–G) IN HUMAN NEUTROPHILS**

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