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17-20 de noviembre de 2021

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Glaucoma is a neurodegenerative disease that affects eye structures and brain areas related to the visual system. Oxidative stress plays a key role in the development and progression of the disease. The aim was to evaluate the redox balance in the cornea in a glaucoma model.
3-month Wistar rats were operated by cauterizing two of the episcleral veins in the left eye: glaucoma group (G n=4); the control group (C n=4) received a sham procedure. Seven days after surgery rats were euthanized, eyes were enucleated and right (RC) and left (LC) corneas were isolated (CICUAL FFyB n° 3314). Damage to macromolecules, antioxidant enzymes activities and NOX and iNOS expression were evaluated.
When compared to CG, GG-LE showed an increase of 100% in protein oxidation (*p<0.01*) and 38% in nitrotyrosine expression (*p<0.05*). Both NOX4 and iNOS expression were 76% (*p<0.01*) and 160 % (*p<0.001*) higher in G-LC, respectively. A 65% increase in SOD activity (*p<0.05*) was measured in both corneas in G. However, GPx activity and CAT levels increased 46% (*p<0.001*) and 80% (*p<0.001*) in G-RC, respectively, without any changes in LC. Finally, there was a 50% decrease in GR activity in G-LC.
These results suggest that glaucoma induces damage to the cornea, such as oxidative modifications to macromolecules, due to an enhancement in oxidative species production from NOX and iNOS. In this context, the RE cornea presents an adaptive response increasing the antioxidant enzymes, while SOD is the only enzyme increased in LC. In addition, glutathione levels could be impaired since its recycling is decreased due to the decay of GR activity in LC. The understanding of corneal impairment in this pathology is important since it could lead to the development of novel therapeutic approaches.

334. (264) **MATERNAL FRUCTOSE CONSUMPTION IMPACTS ON THE DEVELOPMENTAL OUTCOME OF ITS PROGENY**

Marie Lucía Cuervo Sánchez [1], Facundo Prado Spalm [1], Natalia Edith Furland [1], Ana Sofía Vallés[1].
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Recent epidemiological evidence suggests that exposure to maternal obesity increases the risk of neurodevelopmental disorders in offspring. Given the rise in the prevalence of metabolic syndrome (MetS), a clinical condition closely related to obesity, it is important to understand the molecular mechanisms by which maternal MetS might impact offspring behavior and brain function.
Ten, two-months-old female Wistar rats from our colony were separated in two groups of five rats each, control (C) and fructose 20% (F). The former drank tap water while the later drank fructose 20 % (w/v) *ad libitum* during 10 weeks. Both F- and C dams were mated to chow-fed male rats on the 7th week of treatment and maintained on their respective diets throughout pregnancy and sacrificed on day 4 postpartum. At postnatal day (PN) 1, the progeny from both groups were separated from their mothers and continued lactating from control nurse dams. From PN3-PN21 neurodevelopmental reflexes were evaluated. At PN22 all pups were weaned and behavioral tests (open field, marble test, elevated plus maze, novel object recognition, social reciprocal interaction test, tail flick test, rotarod, Kondziela's inverted screen test) were performed on 4- to 12-week-old female and male rats. Results were considered statistically different between the C and the F group when a *p value* of 5% or lower ($p < 0.05$) was obtained when applying the *t-student* test.
Our findings strongly associate maternal fructose consumption with the induction of MetS and infertility. In addition, offspring from the F group presented alterations in the developmental milestones and social behavior; decreased grip strength and increased anxiety. Furthermore, long term memory also showed a tendency to be reduced. No modifications were noted in compulsive like behaviors, locomotion nor in nociception. All in all, maternal fructose consumption impacts on the developmental outcome of its progeny.

335. (310) **OFFPRING FROM MALNOURISHED PARENTS ARE PREDISPOSED TO DEVELOPING METABOLIC DISEASES**

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Growth restriction in utero is associated with the development of obesity and diabetes. The current understanding is that intrauterine deprivation programs the individual for a deprived environment, and that such programming is maladaptive in a no deprived environment. The liver plays an essential role in metabolism regulation.
The objective of this work was to analyze the liver function of the offspring of malnourished parents. Two types of experiments were carried out: 1) the parents (F1) were chronically malnourished from their gestation. They were studied at 120 days of age; 2) other group of malnourished rats at 120 days were mated and their offspring were analyzed at weaning. On day 120 (F1) and day 24 (F2) blood was obteined and the liver was dissected out. Body weight and biochemical parameters were measured. Chronicle protein malnutrition induced increased serum glucose and insulin ($p<0.05$) in F1. Secondly, serum glucose, triglycerides and cholesterol were significantly higher in the malnourished (F2M) group with respect to the control (F2C) and liver proteins and glycogen contein were lower ($p<0.05$). In F2, the values of insulin were higher with respect to the control ($p<0.05$) while the values of leptin and adiponectin were significantly lower in the F2M group. Oxidative stress markers (ROS, lipid peroxidation, and protein carbonylation) showed higher values with respect to the control. These changes were associated with increased pro-inflammatory cytokines production. Serum IL-6, TNF-α and TGF-β were significantly higher in F2M group with respect to the control ($p<0.05$).These results suggest that protein malnutrition during the development predisposes to the occurrence of diabetes and the increment of liver inflammation and oxidative stress markers in their offspring.

336. (314) **EFFECT OF DIETARY SUPPLEMENTATION WITH RESVERATROL, ALPHA-TOCOPHEROL AND PIPERINE ON OXIDATIVE STRESS AND INFLAMMATION IN OLDER ADULTS WITH RISK FACTORS FOR METABOLIC SYNDROME**

Fabiana Lairion[1,2], Raúl Pastor[1], Zulma Manfredi[1], Christian Saporito Magriñá[1,2], Alejandra Cimato[1,2], Isabel Pastor[1], Matías Lasso[1], Aldana Rodriguez[1], Manuela Sorensen[1], Lila López Montañana[1], Margarita Martinez Sarrasague[1,2], Roberto Iermoli[1], Marisa Gabriela Repetto[1,2]
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Oxidative stress, hypertension, blood glucose level and lipid profile are risk factors for metabolic syndrome (MS) and cardiac disease in older adults. The aim of this study is evaluate the protective role of resveratrol supplementation on chronic inflammation and oxidative stress associated to MS. Voluntary patients with a diagnosis of MS (n=92) based on the diagnostic criteria of the National Cholesterol Education Program, Adult treatment Panel III, 2002 received a dietary supplement (RTP: 50 mg resveratrol, 25 mg alpha-tocopherol and 5 mg piperine) along with their usual treatment for a period of 3 months. Piperine increases resveratrol and alpha-tocopherol absorption. Control was the patient himself in baseline conditions, avoiding interindividual variables and bioavailability of active principles. Venous blood was collected from 23 patients (68 ± 5 years), and biochemical markers were assessed in plasma: protein oxidation (measured as carbonyl protein, CO), and interleukin 6 (IL-6); and in red blood cells (RBC): catalase activity. Patients were classified into 3 groups according to the amount of risk factors (blood glucose, HDL cholesterol, triglycerides, waist circumference and blood