CARDIAL ADIPOSE TISSUE: A NEW LINK FOR CARDIOVASCULAR DISEASE?
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Together with the widely known cardiovascular disease (CVD) risk factors, scientific community still look for new ones. Epicardial adipose tissue (EAT) is a visceral adipose tissue surrounding myocardium and coronary arteries, proposed to be an independent risk factor for CVD. The lipidomic study of tissues reveals the role of many bioactive lipids such as ceramides (Cer) on their metabolism. In EAT, these lipids may contribute to CVD risk. We previously demonstrated that Lipoprotein Lipase (LPL) activity is increased in EAT from coronary patients (CAD), probably contributing to an increase in its volume and therefore in CVD. We assessed the lipidome of EAT from CAD as well, demonstrating an enrichment in Cer. In this opportunity, our aim was to evaluate links among EAT LPL, its inhibitor Angiopoietin-like protein 4 (ANGPTL4), its activator glycosylphosphatidylinositol-anchored high density lipoprotein-binding protein-1 (GPIHBP1), and Cer content in the tissue.
Methods and results: in EAT and subcutaneous AT (SAT) from CAD undergoing coronary artery bypass graft (n=25) or valve replacement (No CAD,n=14), LPL activity, ANGPTL4 and GPIHBP1 levels were evaluated by a radiometric assay and western blot, respectively. Tissue lipidomes were evaluated by UHPLC-MS, in positive and negative ionization modes. For statistics, the MetaboAnalyst software was used. In EAT, we found an increase in LPL activity, directly associated with total Cer content (p= 0.002), being the strongest correlation with Cer d18:1/24:1 (p<0.0001). Furthermore, Cer content inversely correlated with ANGPTL4 (p=0.04), but not with GPIHBP1 (p=0.26), although its levels were higher. In EAT, LPL activity was also associated with short and long chain triglycerides, diglycerides and plasmalogens. No associations were observed in SAT.
Conclusion: as a first approach, we propose that the increase in Cer, in particular d18:1/24:1, might be one mediator in the increase of LPL activity, contributing to CVD risk.

315. (064) **MICROENCAPSULATED PEPTIDES FROM BREWER'S SPENT GRAIN HAVE BENEFICIAL EFFECTS UPON LIPID METABOLISM DISORDERS DEVELOPED IN AN EXPERIMENTAL MODEL OF DYSLIPIDEMIA AND INSULIN RESISTANCE**
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Recently, it was demonstrated that peptides obtained from Brewer's spent grain (BSG) - a byproduct of the brewing industry- can exert *in vitro* biofunctional activities including hypolipidemic and antidiabetogenic effects. However, it is unknown whether the bioactive properties remain *in vivo*. The aim of this work was to evaluate the effect of dietary intake of microcapsules containing BSG peptides upon biometric parameters and lipid metabolism disorders in an experimental model of dyslipidemia and insulin resistance. Male Wistar rats were fed from weaning (3-week old) and for 100 days with 1 of 3 randomly assigned experimental diets: a-Control group (C) received a standard commercial rodent diet, b- SRD group received a sucrose-rich diet (SRD), c- SRD+P group was fed with a SRD containing 700 mg/kg body weight/day of BSG protein hydrolysate microencapsulated. We analyzed: a- serum levels of triglycerides (TG), free fatty acids (FFA), cholesterol (Ch), glucose (G) and uric acid, b- biometric parameters-body weight (BW), body length (BL), thoracic (TC) and abdominal circumferences (AC), body mass index (BMI), Lee index- and energy intake, b- visceral adiposity index (VAI), c- In epidydimal adipose tissue (AT): fat cell size, number and lipid content, d-cholesterol esterase (CE) and pancreatic lipase (LP) enzyme activities in cecal contents, e- liver TG and Ch content. Compared with SRD-fed rats SRD+P group shown a significant reduction (P<0.05) of the increased serum lipid levels, hyperglycemia and hyperuricemia. BL, AC, BMI and Lee index were similar in the 3 dietary groups. BW, TC and VAI were increased (P<0.05) in SRD and SRD+P groups, however, a reduction in energy intake and AT adipocytes size and TG content were observed in SRD+P rats. These changes were accompanied by a decrease (P<0.05) of CE and LP enzyme activities and liver lipid content. The results show that BSG protein hydrolysates prevent/ attenuate lipid metabolism disorders developed in SRD-fed rats.

316. (072) **THE CONSUMPTION OF FRIED OIL DURING GROWTH ADVERSELY AFFECTS THE MANDIBULAR BONE FORMATION**
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Dietary lipids play an important role in bone health, which is relevant to develop effective interventions to prevent bone diseases or disorders, especially during the period of growth.The present study evaluated the effects of feeding growing rats with a diet containing fried sunflower oil, on morphometric properties and biomechanical competence of the mandible. **Methods**: Male Wistar rats (21±1 days old) (n=21) were assigned to one of three diet groups: those fed a control diet (C), and a high-fat diet containing either sunflower oil (SFO) or SFO which was repeatedly heated (SFOx). Total body weight and food intake were weekly recorded. After 8 weeks, rats were euthanized. Mandibular growth was estimated with digital calipers on removed bones by taking measurements between anatomical points. Structural properties (load at fracture(Wf), load at yield(Wy), and stiffness(Wydy)), were determined by using a three-point bending test (Instrom 4442). **Results** (mean±SD, ANOVA-SNK): final body weight was lower in SFOx rats as compared with C and SFO groups (310.0±14.7g<339.4±16.6g=336.4±23.0g, respectively; P<0.05). Mandibular weight and length (P=0.001) were negatively affected by high-fat diets rich-SFOx.The deleterious effect of SFOx on mandibular growth was more accentuated on the posterior part (C:11.4±0.3mm= SFO:11.1±0.2mm >SFOx:10.7±0.2mm; P=0.0005). The anterior/posterior ratio indicates that SFOx induced some deformation of the mandible. Structural properties, Wf, Wy and Wydy of the mandible were negatively affected in rats fed SFO either fresh or fried, as compared to the C group (P<0.01). Although Wf and Wy did not show significant differences between SFO and SFOx groups, fried oil intake induced a significant reduction in bone stiffness (Wy/dy). **Conclusion:** The potential adverse effects of consuming SFOx may affect mandibular mass, growth and development. Awarded by UBACyT: 20020170100138BA & 20020170200055BA.

317. (073) **INTAKE OF FRIED SUNFLOWER OIL AND RISK OF HEPATOESTEATOSIS**
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Previous studies by us in growing male rats indicated the prominent role of fried sunflower oil (SFOx) consumption in determining the risk for growth, cardiovascular and bone effects. In this study, we evaluated the effect of SFOx diet on body fat content, visceral fat, hepato-somatic index (HSI) and liver fatty acids (FA) profile of growing rats.

Male weaning Wistar rats (n=21) were randomly assigned to feedings of one of three different diets. The control rats were fed a commercial diet (C) and the experimental rats received either SFO or SFOx diets. SFO was repeatedly heated for a total of 40 hours (SFOx). SFO and SFOx were mixed with commercial rat chow. Rats were fed ad *libitum* throughout the experiment. Total body weight and food consumption were recorded every other day. At eight-week experimental period, body fat content and visceral fat were measured; HSI and liver FA profile (GC,Testo Equipment, Model 270 at 50ºC) were determined. Results. SFOx vs. SFO and C showed that total body weight (323.1±9.1< 335.5±10.9=337.7±8.9 g; p=0.044), % body fat (13.7±0.8b vs.15.9±1.7cvs.10.2±1.3a; p=0.001); % visceral fat (3.55±0.36>3.08±0.38> 2.55±0.33; p=0.008) and HSI (4.17±0.34 vs. 3.64±0.27 =3.66±; p=0.016) were altered. In SFOx rats, serum T-Chol and nonHDL-Chol were the highest (p=0.025 and 0.029, respectively). SFO, SFOx and C groups attained similar serum concentrations of triglycerides and HDL-Chol (P=0.057 and P=0.265, respectively). There were significant higher liver profile of total saturated fatty acids; *trans* FA and unidentified minor compounds in SFOx group (p=0.001, p=0.001 and p=0.025; respectively); meanwhile, SFOx group presented significantly lower in total PUFA (p= 0.001) and MUFA (p=0.001) than SFO and C groups. Conclusions. The potential adverse effects of SFOx denoted the risk for liver dysfunction. Dyslipidemia and increased visceral fat content contribute to the presence and/or progression of hepatosteatosis.

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318. (074) **SIMVASTATIN EXERTS A PLEIOTROPIC EFFECT ON BONE IN RATS UNDER A HYPERCHOLESTEROLEMIC DIET CONSUMPTION**

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There is evidence that lipid and bone metabolisms are mutually regulated. Previously, we had demonstrated the negative effect of high-cholesterol diet(HCD) on bone health. Simvastatin(SMV) blocks cholesterol(chol) biosynthesis in hepatic cells. However, the overall benefits observed with SMV appear to be greater than changes in lipid levels. Indeed, recent studies indicate that statins have beneficial extrahepatic effects. This study evaluated the effects of SMV administration on liver and bone under a hypercholesterolemic diet consumption. **Methods**: 40 rats were assigned to 1 of 4 groups: 1) control(C): fed pellets; 2) fed HCD; 3)C+SMV, fed pellets and SMV was given by gavage(5mg/day); and 4)HCD+SMV. After 5 weeks, rats were euthanized, blood was drawn and chol (mg/dL) and transaminase activities [AST,ALT(UI/l)] were determined. The liver was removed and weighed. The hepato-somatic index (HSI% (organ mass(g)/body mass(g)%)) and Oxidation of 2′,7′ dichlorofluorescein diacetate(DCFH-DA)levels(spectrofluorimetry,u.a./min.mg.prot) were calculated. Structural properties of the femur (load at fracture(Wf), load at yield(Wy), diaphyseal stiffness(Wydy)), were determined (Instrom 4442).

Results (mean±SD,ANOVA-SNK): SMV did not reduce chol (C:63.2±1.1 = C+SMV:67±16.8 < HCD+SMV:214.8±31.2 = HCD:209.3±39.3mg/dL, p<0.001) but improve transaminase activity (p<0.001). In liver, SMV decrease DCFH-DA levels (C 1.87±0.07=C+SMV2.03±0.04 < HCD+SMV3.49±0.26 < HCD4.03±0.1 u.a./min.mg prot, p<0.001). HSI% was higher in HCD groups (p<0.001).However, SMV could revert deleterious effects of HCD on the structural properties of the femoral diaphysis, Wf, Wy and Wy/dy, (p<0.01). Moreover, C+SMV revealed more benefits than expected.

Conclusion: in this study, SMV failed to manage liver and plasma chol concentration, but exhibit “pleiotropic” properties on bone beyond lipid effects. Further clinical evidence is required to evaluate SVM role in the treatment of bone diseases.

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319. (080) **EFFECT OF SOCIAL ISOLATION DUE TO COVID-19 ON BMI IN SCHOOL CHILDREN**

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Introduction: Preventive and compulsory social isolation (ASPO) and the resulting closure of schools and suspension of sports practice reduced physical activity; this could affect children causing changes in lifestyle and consequently body mass index (BMI).

Objective: to evaluate the effect of ASPO (March-October 2020) on the changes in BMI and lifestyle in children aged 6 to 9 years old in La Plata, Buenos Aires.

Methods: Children who underwent their health checks at La Plata Children Hospital-IDIP Health Observatory in the 4 months prior to the ASPO were summoned in November 2020 to assess anthropometric variables (weight, height, BMI expressed as z-score) and lifestyle (diet, physical activity and sleep). A historical control group (GH) was established with anthropometric data from older children who did not experience ASPO between 6 and 9 years old.

Results: 140 children were evaluated. Body weight increased during the ASPO (DzBMI/age: 0.47 vs GH=0.04; p<0.001). Children reported less physical activity (52.3%), increased screen time (75%) and sleeping more hours (46.6%) than before the ASPO. Children with pre-existing overweight/obesity gained more weight (6.25 vs 3.0 kg in normal weight children; p<0.001), associated with reduced physical activity (2 h/day vs 3 h/day in normal weight children; p<0.001) and not to changes in diet.

Conclusion: ASPO affected lifestyle and weight gain in children; those with pre-existing overweight/obesity were more affected. Our findings can guide efforts to preserve and promote child well-being during lockdown, helping governments to decide the confinement rules to apply to children, especially regarding school closing.

320. (090) **CELIAC DISEASE: DAILY ANTIOXIDANT INTAKE FROM FOOD**

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