brain's fatty acids profiles were determined by gas chromatography. Statistical analysis used ANOVA. Results (%Area) were: SERUM: OLEIC O:23.44 ± 3.68a; OS: 18.31 ± 2.22b; C: 10.60 ± 2.01a. LINOLEIC (LA) O: 12.44 ± 1.65b; OS: 12.98 ± 4.31b; C: 18.27 ± 2.81a; LINOLENIC (ALA) O: 0.30 ± 0.09b; OS: 0.32 ± 0.08b; C: 0.92 ± 0.34a; EPA O: 0.65 ± 0.17a; OS: 1.63 ± 0.49b; C: 0.80 ± 0.23a; DHA: O: 1.57 ± 0.58a; OS: 4.00 ± 1.70b; C: 1.33 ± 0.19a. THYMUS: OLEIC O: 21.54 ± 5.92; OS: 24.40 ± 5.04; C: 18.22 ± 3.23. LINOLEIC O: 5.90 ± 0.56b; OS: 6.5 ± 0.61b; C: 10.89 ± 2.18a; ALA O: 0.27 ± 0.02b; OS: 0.30 ± 0.07b; C: 0.49 ± 0.19a; EPA O: 0.49 ± 0.28; OS: 0.50 ± 0.13; C: 0.50 ± 0.12; DHA O: 0.47 ± 0.10a; OS: 0.70 ± 0.12b; C: 0.52 ± 0.16a. BRAIN: OLEIC O: 13.11 ± 2.64; OS: 12.94 ± 1.07; C: 13.14 ± 1.56. LA O: 1.17 ± 0.46; OS: 1.05 ± 0.33; C: 1.26 ± 0.19; ALA O: 0.15 ± 0.03; OS: 0.12 ± 0.04; C: 0.16 ± 0.06; EPA O: 0.46 ± 0,18; OS: 0.38 ± 0.09; C: 0.33 ± 0.07; DHA: O: 11.39 ± 2.04; OS: 11.32 ± 1.69; C: 11.66 ± 1.63. Data with one letter (a,b) in common, were different ($p<0.05$). In sera, O and OS showed lower ALA and LA and higher oleic levels, compared to C. OS presented high levels of EPA and DHA. In thymus, O and OS groups showed lower levels of ALA and LA than C. The OS group only increased DHA. No changes were presented in brain. The results suggest that olive oil exacerbated omega-9 family with diminution of essential fatty acids while organism tries to compensate brain essential fatty acids. Fish oil supplementation increased serum and thymus DHA levels, not modifying low levels of essential fatty acid. Other source of supplementation may be convenient.

0140 - EFFECTS OF PRENATAL STRESS AND POSTNATAL HIGH FAT DIET FEEDING ON BALB/C MICE METABOLISM.

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Abstract/Resumen: In-utero exposure to maternal stress increases short and long term risk of suffering metabolic diseases. Exposure to stressful events leads to an increase in glucocorticoids release by activation of the HPA axis, therefore early programming of the HPA axis has emerged as a key underlying mechanism of stress-related disorders. Evidence suggests that a stressful prenatal environment seems to favour adverse metabolic conditions. To test this hypothesis in BALB/c mice, a strain susceptible to stress but resistant to metabolic effects of a high fat diet (HFD), we exposed female pregnant mice to restraint stress during the last week of pregnancy (2 h/day). Offspring were fed with HFD between weeks 4 and 28 of age. Prenatally stressed (PS) females and males fed with HFD showed higher body weight (females: $p<0.001$, n= 8; males: $p<0.01$, n= 8) and adipose tissue content (adipose tissue weight/body weight, both sexes: $p<0.001$, n= 8). Females were hyperinsulinemic ($p<0.001$, n= 5), with decreased expression of Foxo1 (Forkhead box protein O1) a transcription factor that plays important roles in regulation of gluconeogenesis and glycogenolysis by insulin signaling ($p<0.05$, n= 5) and Adiponectin ($p<0.05$, n= 5) in adipose tissue. On the other hand, PS males (fed with standard or HFD) had hypertriglyceridemia ($p<0.001$, n= 8) and hypercholesterolemia ($p<0.001$, n= 8). PS per se, in males, decreased the expression of Adiponectin ($p<0.01$, n= 5). PS animals showed a great susceptibility to develop obesity. We conclude that PS may give rise to some adverse effects, and abnormal phenotype may be provoked by or exacerbated in a later life nutritional challenge. We intend to continue our research by evaluating whether epigenetic alterations are responsible for the observed gene expression alterations.

0144 - MECHANISMS UNDERLYING SKELETAL MUSCLE LIPOTOXICITY IN DYSLIPEMIC INSULIN-RESISTANT RATS: EFFECTS OF DIETARY CHIA (SALVIA HISPANICA L.) SEED.

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LABORATORIO DE ESTUDIO DE ENFERMEDADES METABÓLICAS RELACIONADAS CON LA NUTRICIÓN- FBCB-UNL

Abstract/Resumen: Lipid accretion in skeletal muscle (SM) is related to the development of lipotoxicity and insulin resistance (IR), however, the mechanisms involved are not fully clarified. We previously shown that rats fed a sucrose-rich diet (SRD) develops IR, dyslipidemia, and SM lipid accretion. Moreover, we demonstrated that all of them were reversed when chia seed (Salvia hispanica L.)- rich in alpha-linolenic acid (ALA, 18:3 n-3)- was administered as a dietary source of fat in SRD-fed rats. The aims of this study were: (i) to explore the mechanisms underlying SM lipotoxicity in SRD-fed rats (ii) to investigate the effects of chia seed on these mechanisms. Male Wistar rats were fed a SRD for 3 months. Half of the animals continued with the SRD until month 6, the other half was fed a SRD in which the fat source, corn oil, was replaced by chia seed from month 3 to 6 (SRD+chia). Another group consumed a reference diet all the time. In SM we analyzed: a. muscle-type carnitine palmitoyltransferase (M-CPT), fatty acid synthase (FAS), and glucose-6-phosphate dehydrogenase (G-6-PDH) enzyme activities, b. protein mass levels of PPARalpha, PPARgamma, total AMPK, pAMPK, precursor and mature forms of SREBP-1 and sarcolemmal FAT/CD36, c. fatty acid composition of SM phospholipids. SM of SRD-fed rats showed a significant reduction ($p<0.05$) of M-CPT 1 enzyme activity, PPARs and pAMPK protein levels. FAS, G-6-PDH enzyme activities, the mature form of SREBP-1 and FAT/CD 36 were increased ($p<0.05$). In SRD+chia-fed rats M-CPT 1 enzyme activity, PPARs and pAMPK protein levels were normalized ($p<0.05$). The precursor and mature forms of SREBP-1 and lipogenic enzyme activities were decreased ($p<0.05$). FAT/CD36 and n-3/n-6 fatty acids ratio of membrane phospholipids were increased ($p<0.05$). In summary, this study shows some mechanisms involved in SM lipotoxicity of insulin-resistant rats fed a SRD and provides novel information on the beneficial effects of chia seed on these mechanisms.

0207 - ALPHA-LINOLENIC ACID-RICH CHIA (SALVIA HISPANICA L.) SEED AMELIORATES ADIPOSE TISSUE DYSFUNTION IN AN EXPERIMENTAL MODEL OF VISCERAL ADIPOSITY AND INSULIN RESISTANCE BY MODULATING LIPID METABOLISM.

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Abstract/Resumen: Salvia hispanica L. (chia) seed is one of the richest botanical sources of alpha;-linolenic acid (ALA, 18:3 n-3)- and it has generated considerable research interest in recent years. We previously shown in dyslipemic insulin resistant rats fed a sucrose-rich diet (SRD), which have visceral adiposity, that the replacement of corn oil by chia seed in the SRD reduces epididimal adipocyte hypertrophy, triglyceride content, lipogenic enzyme activities and lipolysis. This study aimed to further explore if changes in adipocyte lipid metabolism could be involved in the beneficial effect of chia seed on reducing visceral adiposity. Male Wistar rats were fed a SRD for 3 months. Half of the animals continued with the SRD until month 6, the other half was fed a SRD in which the fat source, corn oil, was replaced by chia seed from month 3 to 6 (SRD+chia). Another group consumed a reference diet all the time. We analyzed: a. morphometrical parameters -body weight (BW), body length, thoracic (TC) and abdominal circunference (AC), body mass