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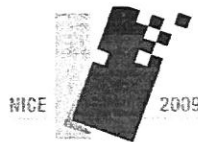
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ABSTRACT BOOK

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BODY FAT DISTRIBUTION DETERMINED WITH DXA IN WOMEN WITH PCOS

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Most women with PCOS are characterized with upper body obesity. The aim of this study was to perform DXA examination in order to evaluate body fat distribution in 20 PCOS that were matched by their body mass index (BMI) and age with control group (C) of 20 women.

Methods: DXA assessment was performed with Lunar DPX-NT system. Total fat mass (TFM), trunkal fat mass (TRFM), android fat mass (AFM) and gynoid fat mass (GFM) were determined, as well as centrality index (CI) as a ratio between the android fat mass percentage (AFM%) and gynoid fat mass percentage (GFM%).

Results: BMI values in PCOS were $29.36 \pm 5.58 \text{ kg/m}^2$, not significantly different compared to C ($29.09 \pm 5.36 \text{ kg/m}^2$). TFM ($35.12 \pm 11.15 \text{ kg}$) and TRFM ($18.21 \pm 5.46 \text{ kg}$) in PCOS were also not significantly different compared to TFM ($31.5 \pm 10.23 \text{ kg}$) and TRFM ($15.64 \pm 5.57 \text{ kg}$) in C. Android fat mass percentage in PCOS ($53.81 \pm 7.78\%$) was significantly higher compared to C ($44.82 \pm 9.33\%$) ($p < 0.01$). Gynoid fat mass percentage in PCOS ($52.75 \pm 6.76\%$) did not differ compared to correspondent values in C ($52.91 \pm 4.72\%$). CI in PCOS ($1.02 \pm 0.08\%$) was significantly higher compared to C ($0.84 \pm 0.12\%$) ($p < 0.0001$).

Conclusions: PCOS women were characterized with increased android fat mass percentage and centrality index, indicating abdominal fat distribution. Centrality index increase was a result of higher android fat mass percentage, but not significant different gynoid fat mass percentage in PCOS women compared to controls. Trunk fat mass and total fat mass were not different among the groups confirming redistribution of the fat mass in central depots.

LIVER FUNCTION TEST DURING METFORMIN THERAPY IN PATIENTS WITH POLYCYSTIC OVARY SYNDROME (PCOS)

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Background: PCOS shares several features of Metabolic Syndrome with nonalcoholic fatty liver disease (NAFLD). To evaluate the prevalence of NAFLD and the effect of standard therapy with Metformin on liver function in patients with PCOS and hyperinsulinaemia.

Patients and methods: We enrolled 50 non-diabetic patients with PCOS. NAFLD was assessed by ultrasound (US). Anthropometric variables, OGTT, serum lipids and aminotransferases, and HOMA index were determined at basal visit and after 6 months of therapy with Metformin. The Wilcoxon paired test was used to assess the modification after treatment, $p=0.05$ was considered significant.

Results: 13 patients (age: 25.69 ± 7.38 years; BMI: 30.96 ± 8.23) completed the treatment. At the entry basal visit 30.8% (4/13) showed MS, 10/13 (76.9%) had NAFLD. Subjects with PCOS and NAFLD were younger than subjects without NAFLD (24.10 ± 6.84 VS 31 ± 7.81) with higher waist-hip-ratio ($p < 0.05$). After 6 months of Metformin therapy the AUC was significantly improved in all subject ($p: 0.002$), HDL-Cholesterol significantly increased ($p < 0.05$) and the AST/ALT ratio was significantly improved (0.90 ± 0.39 vs 0.72 ± 0.32 , $p: 0.028$) even if a significant reduction of the ultrasound degree of steatosis was not observed.

Discussion: Six months therapy with Metformin in patients with PCOS was able to improve severity of liver disease assessed by AST/ALT ratio even if we did not observed a significant modification of steatosis at US. The reduction of IR incidence and the increase the HDL Cholesterol levels could

lead to a reduction of cardiovascular risk and improving liver function tests in women with PCOS.

MITOCHONDRIAL FUNCTION IN LAMB AS A CONSEQUENCE OF MATERNAL CALORIC RESTRICTION DURING PREGNANCY AND HIGH-FAT-HIGH-CARBOHYDRATE DIET POST PARTEM

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Aim: To examine effects of foetal programming and catch-up growth upon adult life. We examined lambs of ewes that were exposed to a low calorie (L) diet containing only 50% of their normal calorie intake. Subsequently half of the lambs were exposed to a high-fat high-carbohydrate (HFHC) diet before half of the lambs were sacrificed at the age of 6 month and half lived on till 2 years of age on a normal diet.

Methods: Oxygen consumption was measured in preparations of skinned muscle fibres using the Oroboros oxygen electrodes. mRNA expression levels of selected genes was examined through real-time RT-PCR.

Results: The HFHC diet induced a 50% increase in mtDNA and in mitochondrial $\text{VO}_{2\text{-max}}$. The most pronounced change, however, was a two-fold change in respiratory coupling ratio (RCR) in the group receiving HFHC post partem, independent of the feeding of the mothers. UCP3 mRNA levels were decreased in groups fed HFHC diet compared to control. PGC-1 α mRNA levels were increased in the L group independent of HFHC. After 2 years $\text{VO}_{2\text{-max}}$ was decreased.

Conclusion: The increased mitochondrial coupling induced by HFHC feeding will contribute to an increased ROS load and thereby offer a possible mechanism of how such combined effects of intrauterine and postnatal nutritional conditions may damage mitochondria and suggest a mechanism that further down the road may lead to metabolic disorders and type 2 diabetes.

RISK FACTORS FOR GESTATIONAL DIABETES MELLITUS: SIMILARITIES WITH TYPE 2 DIABETES

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Gestational diabetes mellitus (GDM) is an important risk factor for type 2 diabetes (T2D).

Aims: To identify features measured before and during pregnancy that may be associated with an increased risk for GDM.

Methods: Medical records of 163 women with GDM and 987 women without GDM who gave birth at the Centre Mère-Enfant, Laval University Health Centre (Quebec City, Canada) in 2007 were retrospectively reviewed. Odds ratio adjusted for age and pre-pregnancy BMI (aOR) were computed.

Results: Compared to women without GDM, women with GDM were older (31.1 ± 5.1 vs. 29.0 ± 4.5 yrs, $p < 0.0001$), had a higher pre-pregnancy BMI (29.0 ± 7.7 vs. $24.1 \pm 5.2 \text{ kg/m}^2$, $p < 0.0001$) and higher 60 minutes-post 50g OGTT values (9.82 ± 1.61 vs. $6.14 \pm 1.37 \text{ mmol/L}$, $p < 0.0001$). Women with GDM were more likely to have a family history of diabetes (aOR, 95%CI: