P-IS-65 Preliminary Study for Quantification of Mitochondrial DNA (mtDNA) on the Semen of Infertile Men.

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[Objective] Mitochondria are important organelle that provide energy for the survival of cells. The purpose of this study is to assess the quantity of mtDNA in the semen of infertile men. [Methods] We used 4 semen samples obtained from patients who gave informed consent in accordance with the rules of the institutional ethics committee. The spermatozoa count from 4 patients (2 with normozoospermia, 2 with oligozoospermia) was as follows: 130×, 80×, 15×, and 10× ; 10^6/ml. We extracted mtDNA from the samples by treating them with Dithiothreitol (DTT) and Proteinase K. Then mtDNA was amplified using long and accurate (LA) PCR primers. Also in order to compare the copy number of each sample, we amplified the template for 40 cycles by Real-time PCR. [Results] All mtDNA from semen samples could be amplified. The number of cycles to amplify each sample was 16, 20 (normozoospermia), 16, and 19 (oligozoospermia). There were no significant differences in cycle numbers between each sample. [Conclusion] Mitochondria supply ATP energy to cells through oxidative phosphorylation. In this study did not find any significant difference in the number of the copies of mtDNA between patients with normozoospermia and oligozoospermia. There is a possibility that patients with oligozoospermia, even if they have a sufficient number of normal mtDNA, have a lot of mutated mtDNA.

P-IS-66 Differential responds of prolactin secretion to hyperglycaemia and hypoglycaemia in healthy women and in women with polycystic ovary syndrome

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Introduction : Polycystic ovary syndrome (PCOS) is associated with insulin resistance and hyperinsulinemia. The aim of this study was to determine the influence of the serum glucose concentration on prolactin secretion in healthy women and in PCOS patients and to analyse whether the set-point of glucose concentration at which changes occur is altered in PCOS patients.
Material and Methods : 8 women with PCOS and 20 healthy controls underwent a stepwise hyperglycaemic to hypoglycaemic hyperinsulinemic clamp experiment Patients were infused with insulin at a rate of 2,5 mU/min-1 x kg-1. After a hyperglycaemic state was obtained at a level of 8,8 mmol/l and maintained for 30 minutes the plasma glucose concentration was decreased stepwise. The plasma glucose was held constant for 30 minutes at a concentrations of 6,8 mmol/l, 4,8 mmol/l and 2,8 mmol/l. Blood was drawn every 15 minutes resulting in 3 prolactin levels on each glucose plateau of which the mean value was calculated.
Results : Fasting prolactin concentrations were significantly lower in PCOS patients compared to controls (10.3 vs. 16.3 ,$p < 0.015$). In controls prolactin levels decreased significantly by 23.8%compared to baseline during hyperglycaemia ($p = 0.045$). With decreasing glucose concentrations prolactin levels increased gradually until reaching baseline levels at a glucose concentration of 2,8 mmol/l. In women with PCOS prolactin secretion remained at baseline levels during hyperglycaemia, while in increased significantly during hypoglycaemia reaching 151.2%of the baseline values at a glucose concentration of 2,8 mmol/l($p = 0.002$). PCOS patients had a higher body mass index(28.2 · 6.3vs. 22.6 · 2.5, $p = 0.06$), which was not statistically significant, but a significantly higher waist-to-hip-ratio(0.79 · 0.9 vs. 0.74 · 0.5, $p = 0.033$) compared to control patients. There were no differences in serum lipid levels and in the fasting glucose concentration.
Conclusion : In PCOS patients the glucose sensor seems to be altered. The decrease of prolactin secretion during hyperglycaemia observed in healthy controls is missing in PCOS patients. Even non-insulin resistant PCOS patients with normal fasting glucose concentrations might be adapted to hyperglycaemia. The hypoglycaemic state increases the prolactin secretion compared to fasting levels in PCOS patients. This reaction resembles the phenomena observed in type 2 diabetes as counterregulation to hypoglycaemia begins at normoglycaemic ranges in poorly controlled type 2 diabetes.