J Diabetes Metab 2016, 7:8(Suppl)
http://dx.doi.org/10.4172/2155-6156.C1.052

12th European Diabetes Congress

September 15-17, 2016 Berlin, Germany

Early diagnosis, incretin-based therapies and gene therapy approaches for type 2 diabetes

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It has now become obvious that the pathophysiological defects leading to type 2 diabetes (T2D) is much more complex than thought before. Insulin resistance is an early event in the course of T2D development. The transition from normoglycemy to pre-diabetes is usually a gradual phenomenon that occurs over 5-10 years during which the disease remains undetected. Among the routinely practiced T2D screening criteria, like, FPG, IFG, IGT or HbA1c, still the issue of a preferable one is debated. Here I present more precise noninvasive pre-symptomatic diagnosis and risk assessment strategies including non-coding RNAs signature in peripheral blood. Life style changes with addition of metformin, sulphonylureas, glinides, α-glucosidase inhibitors, thiazolidinediones and/or exogenous insulin are recommended as the present treatment options. These treatments offer improvement in glycemic control, but in many instances produce significant adverse side effects. Various novel incretin-based therapies like prolonging GLP-1 receptor agonists action, orally GLP-1 receptor agonists, GLP-1 secretion by activating GLP-1-producing intestinal L-cells, synthetic engineered peptides as co-agonists stimulating more than one receptor, etc. are discussed here. I also present our experiences regarding development of successful gene therapy using intestinal K-cells which are specialized for GIP production. Engineering these cells to produce insulin in response to the ingested carbohydrates successfully achieved. Oral gene delivery to these cells using nanoparticles with appropriate protective coats as well as plant exosomal gene delivery to the stem cell precursors of K-cells located at the base of intestinal crypts resulted in long lasting insulin expression by gut K-cells and pronounced treatment of T2D.

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Study of possible relation between maternal serum resistin and insulin resistance in patients with pre-eclampsia

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Introduction: In humans resistin antagonizes the effects of insulin on glucose metabolism in liver and skeletal muscle, interacts with and reinforces inflammatory pathways and may promote endothelial cell activation. Increased resistin levels have been associated with obesity, insulin resistance, metabolic syndrome, type 2 diabetes and increased cardiovascular risk

Objectives: Our study aimed to investigate the utility of maternal serum resistin in women with preeclampsia compared to normal pregnant women and its relation to insulin resistance.

Methods: The study was conducted on ninety (90) females, divided into two groups: Group I: Pre-eclampsia (n=60), Group II: Healthy pregnant and Control (n=30). All individuals were subjected to the following after an informed oral and written consent: Full history taking, clinical examination with special emphasis on edema, blood pressure measurement and Maternal body mass index (BMI); Index (weight (kg) / height^2 (m^2)), Determination of gestational age according to the date of the last menstrual period and confirmed by first trimester ultrasound laboratory investigations including CBC, AST, ALT, BUN, creatinine, HOMA-IR and serum resistin.

Results: Statistical comparison between pre-eclamptic patients (Group I), and the healthy control group (Group II) regarding the different studied parameters revealed a highly statistically significant increase in the patients group than the control group regarding SBP, DBP, BMI, CRE, AST, ALT, 50 g oral glucose challenge test (GCT), FBG, fasting insulin, HOMAIR and resistin. On the contrary, there was a highly statistically significant decrease in the patients group than the control group regarding HB.

Conclusion: In this study it was found that elevated serum resistin levels could be associated with exaggerated insulin resistance in patients with preeclampsia. Further studies are needed to clarify the role of resistin in the patho-physiology of preeclampsia and insulin resistance.

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