CHAPTER 33

MEDICAL SCIENCES
MEDICINE

Doctoral Theses

01. MISHRA (Brijesh Kumar)
Study of Polymorphism of Genes Associated with Postprandial Lipemia and their Expression in Adipose Tissue in Patients with Type 2 Daibetes Mellitus.
Supervisor : Dr. S. V. Madhu
Th 24211

Abstract
(Not Verified)

Aim: To ascertain whether polymorphisms of various genes known to be associated with postprandial lipaemia or their altered adipocyte expression is associated with insulin resistance and glucose intolerance. Methodology: The present study compared postprandial lipaemic responses to oral fat challenge and polymorphisms of PPL genes in 200 in age, sex and BMI matched subjects consisting of equal number of those with T2DM, Prediabetes and NGT. Standard oral fat challenge test was performed in all subjects and blood samples (fasting and postprandial at 2,4,6 and 8hrs) were collected for biochemical and genetic analysis of PPL associated genes. In 30 subjects (10 each in T2DM, Pre-diabetes and NGT group) who underwent abdominal surgery, visceral adipose tissue and subcutaneous adipose tissues were collected for gene expression studies. Results: There was significantly higher postprandial Tg levels in T2DM group as compared to prediabetes and NGT groups. Of the 17 postprandial lipemia associated genes studied, polymorphisms of TCF7L2, PPARϒ, ADIPOQ, APOE, CETP, APOA5, IL-6, GCKR and PRDM-16 showed significant association with risk of T2DM/Prediabetes. There was significant association of Polymorphisms of TCF7L2, ADIPOQ and IL6 genes with postprandial Tg levels along with glycaemic and insulin resistance parameters. Conclusion: The study found significant association of polymorphisms and altered adipose tissue expression of 4 ppl associated genes (TCF7L2,PPARi¡,ADIPOQ,IL-6) with risk of diabetes/prediabetes, postprandial hypertriglyceridemia and parameters of glycaemia and insulin resistance. It would thus appear that there is a strong genetic basis of development of ppl as well as its link with diabetes risk.

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02. DAR (Sadaf Bashir)
Study of Micrornas in HBX-Induced Hepatocellular Carcinoma
Supervisor : Dr. Richa Dewan
Th 24207

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carcinoma 5B. HBX regulates microrana-21 to stimulate cell proliferation through akt pathway by targeting programmed cell death protein (PDCD4) and phosphatase tensin homologue (PTEN) in HBV-associated hepatocellular carcinoma 5C. HBX protein governs microrna-221 upregulation to promote aberrent proliferation by targeting P57 and P27 ecpression in human HBV modulated hepatocellular carcinoma. Discussion. Summary and conclusion. Limitations and future perspective of the study. References. Appendix.