UGC MINOR RESEARCH PROJECT Entitled

" Evaluation of the interrelationship between thyroid function and metabolic

syndrome in suitable animal models"

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SUMMARY OF THE PROJECT REPORT

Project Title: Evaluation of the interrelationship between thyroid function and metabolic syndrome in suitable animal models

Metabolic syndrome is a major public health burden affecting about 20-30% of adult population worldwide and its prevalence in South Asians, particularly Indians is on the steady increase. There is indication that metabolic syndrome modulate thyroid function by increasing TRH output in normal conditions. In the present project proposal, the interrelationship between metabolic syndrome and thyroid function was investigated in an animal model since no systematic study has been performed on the thyroid functions in suitable animal models during the emergence of the metabolic syndrome. HFSC diet significantly (p<0.05) impaired fasting blood glucose at 0 minute in the test as compared to the control after 1 month of feeding which normalized at the end of 120 minutes. After 4 and 5 months of feeding, significantly (p < 0.01and p < 0.001) higher impaired glucose tolerance was observed till 120 minutes of feeding with the glucose levels shooting very high in the test as compared to the control. A corresponding HOMA-IR analysis revealed that the HOMA-IR levels of the test mice were higher than the control within 1 month and significantly higher after 3 months (p < 0.001) and 5 months (p < 0.001) 0.001) of feeding. The insulin analysis supports the hypothesis of hepatic and muscle insulin resistance. Thus, impaired glucose tolerance is an intermediate phase between Normal Glucose Tolerance and Type 2 Diabetes. In our study, the test Leptin levels were found to be lower in the test although not significantly after a month of feeding as compared to the control indicating that the body's sensitivity to leptin has not been lost yet. However, the test leptin levels were significantly (p<0.05) higher than the control after feeding for a period of 5 months. However, in our study insulin secretion as stated earlier is higher in test than control indicating loss of regulation. TSH levels start to rise by third month and were found to be higher in the test as compared to control in the fifth month, indicating loss of regulation (Fig 1). Triiodothyronine levels start to rise by third month and were found to be higher in the test as compared to control in the fifth month, indicating loss of regulation. Thus our study shows that HFSC diet causes glucose intolerance, hyperleptinemia, hyperinsulinemia in the test. HFSC diet was also found to impair pituitary hormone like TSH after 3 months after 5 months post feeding. Histology of thyroid gland shows a prominent difference in the test group compared with control group with respect to follicular epithelium and colloid containing cells. This shows that metabolic syndrome has effect even on the histology of thyroid gland which is the reason for impairment of thyroid stimulating hormone and triiodothyronine.

Contributions to the society:

- Metabolic syndrome is a set of complications with hyperinsulinemia, hyperleptinemia and also thyroid disorders. But the molecular mechanism for the interrelationships between the metabolic syndrome and thyroid hormone was derived from this project.
- Relationship between diabetes, glucose intolerance and thyroid disorders was derived from this project.
- Variations in thyroid hormones and histopathology of thyroid gland in metabolic syndrome induction subjects were very well studied at molecular level.