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PhD Dissertation Defense

Entitled

Effects of Adropin on Metabolic Parameters, Oxidative Stress, and Cell Proliferation in Animal Model of Diabetes Mellitus

by

Ifrah Ali

Faculty Advisor

Prof. Ernest Adeghate, Department of Anatomy

College of Medicine & Health Sciences (CMHS)

Date & Venue

11:00 am

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Yannah Theater, Second Floor, Block C (2C010), Male Side, CMHS

Online

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Abstract

Diabetes mellitus (DM) is a major health issue that has reached alarming levels, affecting around 537 million of the global adult population. DM is a group of metabolic diseases characterized by hyperglycemia with disturbance in carbohydrate, fat, and protein metabolism. The proposed therapeutic options for DM are different according to their mechanism of action, and peptide therapy is one of these options. Adropin, a peptide hormone involved in energy homeostasis, has shown promising results in the treatment of diabetes by lowering fasting blood glucose, reducing insulin resistance, and minimizing fat storage. Moreover, it has been demonstrated that adropin is engaged in other biological activities such as reducing oxidative stress and inflammation. However, little is known about the ability of adropin to modulate pancreatic peptide secretion in DM. So, the aim of this study was to investigate the role of adropin in controlling glucose levels by enhancing pancreatic peptide secretion in streptozotocin-induced diabetic rats. To achieve our aim, normal and diabetic Wistar rats were treated with adropin (2.1 µg/kg/day) for a period of 10 days. Fasting blood glucose and body weight were measured. Pancreatic tissue samples were collected for histomorphological analysis and blood was collected for oxidative stress assay and peptide analysis. We reported enhancement in glucose utilization, decrease in □-cells number as well as reduction in glucagon release in adropin-treated diabetic rats. Furthermore, adropin significantly augmented the secretion of certain endocrine pancreatic hormones including C-peptide, amylin, and Glucagon Like peptide-1 (GLP-1) in rats with DM. Interestingly, adropin significantly increased glutathione reductase expression in the pancreas tissue, in addition to serum total glutathione in the diabetic treated rats compare to diabetic untreated. We found that diabetes induction stimulated cell proliferation in both exocrine and endocrine pancreas, and adropin dramatically attenuated this effect in pancreatic exocrine, but not in the islet of Langerhans. This work will add value to the field of diabetes by providing better understanding of how adropin is involved in maintaining glucose homeostasis and glucagon secretion.

Keywords: Diabetes mellitus, Adropin, Glucose homeostasis, Endocrine pancreas, Exocrine pancreas, Rats.