

EFFECT OF EXERCISE ON SOMATOSTATIN C-PEPTIDE, AND FRUCTOSAMINE IN BOTH INSULIN DEPENDENT AND NON INSULIN DEPENDENT DIABETICS



A THESIS
Submitted For Partial Fulfillment of the
Requirement For The Degree of M.D. In
General Medicine

BY

Magda Shukry Mohamed
M.B., B.Ch - M.S In General Medicine
Ain Shams University

Supervisors

Prof. Dr.

Samir M. Sadek
Prof. of General Medicine and Endocrinology

Prof. Dr.

M. Amin Fikry
Prof. of General Medicine and Endocrinology

Prof. Dr.

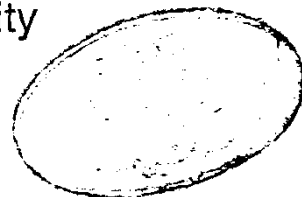
Nadia Abd-El-Salam
Prof. of Physical Medicine

Prof. Dr.

M. Fahmy Abd-El-Aziz
Assistant Prof. of General Medicine

Faculty of Medicine
Ain Shams University

1993



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TO MY FAMILY

&

*TO THE MEMORY OF
MY FATHER
AND
MY BROTHER*



ACKNOWLEDGEMENT

I wish to express my deepest gratitude and appreciation to Prof. Dr. Samir Mohamed Sadek, Professor of General Medicine and Head of Endocrinology Department, Ain Shams University, for his consistent supervision, valuable suggestions, and revising all the details of this work. His fatherly attitude was a great help to complete this work.

Sincere thanks are to Prof. Dr. Mohamed Amin Fikry, Professor of General Medicine, for his kind supervision, encouragement, generous help, and valuable suggestions.

My appreciation to Prof. Dr. Nadia Abd El-Salam, Professor and head of Physical Medicine Department, for her kind supervision and valuable advices.

Thanks are also due to Dr. Mohamed Fahmy Abd El-Aziz, Assistant Professor of General Medicine, for his kind help, encouragement and patient guidance.

I also would like to thank Dr. Salah Shelbaia, Assistant Professor of General Medicine, for his kind help, and continuous encouragement.

great thanks to my dear colleagues, the chemists Tahany Mohamed and Laila Aziz, Staff of the Endocrinology Laboratory, our patients and every one participated, in some way or another, in making this work feasible.

CONTENTS

INTRODUCTION	(1)
AIM OF THE WORK	(3)
REVIEW OF LITERATURE	
* Glucose Homeostasis	(4)
* Diabetes Mellitus	(45)
* Non enzymatic Glycation of Proteins	(58)
* Somatostatin	(63)
* Exercise and Diabetes Mellitus	(105)
SUBJECTS AND METHODS	(154)
RESULTS	(169)
DISCUSSION	(242)
SUMMARY AND CONCLUSION	(271)
RECOMMENDATIONS	(276)
REFERENCES	(277)
ARABIC SUMMARY.	

INTRODUCTION

INTRODUCTION

Since the early demonstration of its glucose-lowering effect (Lawrence, 1926), exercise has been considered an important component of the treatment of diabetes mellitus. As a result, standard medical textbooks and diabetes teaching programs have encouraged increased physical activity for patients with diabetes (Zinman and Vranic, 1985).

In normal subjects, both physical fitness and acute exercise per se are associated with lower-than normal fasting and stimulated insulin levels (Wirth et al., 1987). This reduction of insulin levels is likely to be due to reduced insulin secretion rather than more rapid clearance, as suggested by the finding of diminished insulin released from the isolated islets of physically trained rats (Galbo et al., 1981).

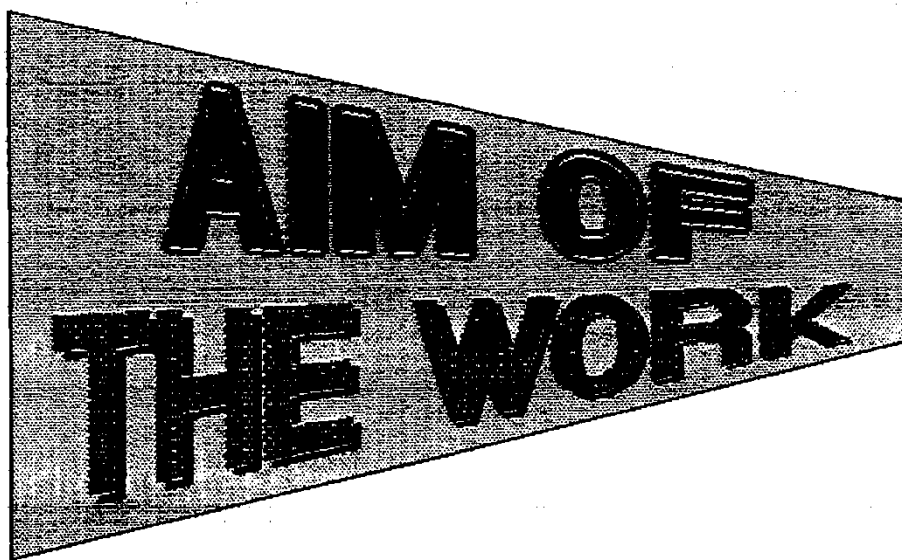
As regards diabetes mellitus, mild physical exercise results in a fall of blood glucose levels in controlled diabetic patients, this acute effect of exercise might be used to inhibit the rise of blood glucose seen in insulin treated diabetics after food intake. In contrast, physical



activity will further deteriorate the metabolic situation in decompensated diabetic patients by an increase in blood glucose and a rapid aggravation of ketosis (Berger and Berchtold, 1979).

Glucose homeostasis is known to be controlled not only by insulin, but also by the counter regulatory hormones including glucagon, cortisol, catecholamines and growth hormone. Physiologically, somatostatin suppresses insulin, glucagon and growth hormone release (Reichlin, 1983a).

Wasserman et al. (1984) found that glucagon suppression induced by somatostatin infusion results in hypoglycemia during exercise and rest. However, few studies were done on the effect of exercise on endogenous somatostatin or to demonstrate the role of endogenous somatostatin in glucose homeostasis during exercise.



AIM OF THE WORK

The aim of this work is:

1) To estimate the levels of somatostatin and c-peptide before and after exercise in both insulin-dependent and non-insulin-dependent diabetics in order to demonstrate the effect of exercise on their release and to show the role of changes in insulin and somatostatin in controlling glucose homeostasis in diabetics during exercise.

2) To study the effect of exercise on fasting blood glucose and fructosamine levels in both types of diabetes mellitus in order to show the beneficial effects of exercise on diabetic control.

REVIEW OF LITERATURE

GLUCOSE HOMEOSTASIS

Maintenance of the plasma glucose concentration is critical to survival, because plasma glucose is the predominant metabolic fuel utilized by the central nervous system under most conditions. The central nervous system can not synthesize glucose, store more than a few minutes supply, or concentrate glucose from the circulation. Thus, brief hypoglycemia can cause profound brain dysfunction, and prolonged, severe hypoglycemia causes brain death. It is, therefore, not surprising that glucoregulatory systems have evolved to prevent or correct hypoglycemia (Cryer, 1985).

The normal fasting level of glucose in peripheral venous blood is 70-110 mg/dl (3.9-5.6 mmol/L). In arterial blood, the glucose level is 15-30 mg/dl higher than in venous blood (Ganong, 1991).

The plasma glucose concentration is the result of glucose influx to the circulation and glucose efflux from the circulation. Glucose entry into the circulation results from: (1) the absorption of glucose derived from digested dietary carbohydrate; (2) the release of endogenous glucose from the liver. Endogenous glucose is derived from hepatic

glycogen (glycogenolysis) and glucose newly synthesized in the liver (gluconeogenesis) (Cryer, 1984).

Glucose exits from the circulation into a variety of tissues where it is rapidly metabolized. For practical purposes, free glucose does not exist in tissues. Basically, glucose has the following potential fates: (1) Storage in the form of glycogen (2) glycolysis to pyruvate that can be; (a) after conversion to lactate, recycled to form glucose in the liver; (b) utilized as a metabolic fuel by oxidation to carbon dioxide and water; or (c) converted to acetyl CoA and hence fatty acids and stored in the form of triglycerides (Cryer, 1984). Fig. (1).

The liver is for practical purposes the sole source of endogenous glucose production. Renal gluconeogenesis and glucose release contribute substantially to the systemic glucose pool only during prolonged starvation. Under conditions of high glucose output, the energy needs of the liver are largely provided by the β -oxidation of fatty acids. On the other hand, the liver can also be an organ of net glucose uptake with storage as glycogen, oxidation for energy, and conversion to fat, which either can remain in the liver or be transported to other tissues as very low