# EFFECT OF UPPER VERSUS LOWER LIMB EXERCISES ON BLOOD GLUCOSE LEVEL IN TYPE 2 DIABETIC PATIENTS

#### **Thesis**

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By

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### **Abstract**

This study was conducted to investigate the effect of upper versus lower limbs exercises on blood glucose level in type 2 diabetic patients. Forty sedentary subjects from both sex (20male and 20 female) their age ranged from 40 to 60 years, onset of disease ranged from 3 to 6 years with body mass index ranged from (30 to 34.9) selected from the diabetes out clinic in Al Mataria hospital. All patients have hyperglycemia tested by a two hours plasma glucose ≥200 mg/dl (11.1mmol/l) during an OGTT. The test should be performed as described by the World Health Organization, using a glucose load containing the equivalent of 75-g anhydrous glucose dissolved in water. Patients assigned into two groups, group (A) upper limb group and group (B) lower limb group, each group will be 20 patients from both sex (10 male and 10 female) for each group .Performing exercises for 30 minutes and within 75% to 85% of their maximum heart rate. The blood sample was taken from antecupital vein in 2hours postprandial state immediately before exercises and after performing exercises. There was significant difference in post treatment values between both groups. The percentage of decrease in blood glucose level between pre and post exercise training in upper limb group (A) was 12.82% were the percentage of decrease in blood glucose level between pre and post exercise training in lower limb group (B) was 6.31%.

**Key words:** Acute exercise, Blood glucose, Postprandial, Type 2 diabetes.

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### List of Abbreviation

• ACTH Adrenocorticotropic hormone

• AGEs Advanced glycation endproducts

• AICAR Adenosine analog 5-aminoimidazole-4-carboxamide-

1-ß -D-ribofuranoside

• AIDS Acquired Immune Deficiency Syndrome

AMPK Adinosine monophosphate kinase

ATP Adenosine triphosphateA1C Glycosylated hemoglobin

• **BOP** Bleeding on probing

• **BP** Blood pressure

CAD Coronary artery disease

CaM Calmodulin

CBG casual blood glucose

CHO Carbohydrate

CVD Cardiovascular diseaseDKA Diabetic ketoacidosis

• **DPN** Distal symmetric polyneuropathy

• **DPP** Diabetes Prevention Program

• ECG Electrocardiogram

• **EGP** Endogenous glucose production

ESRD End stage renal diseaseFPG Fasting plasma glucose

GAD Glutamic acid decarboxylase
 GDM Gestational diabetes mellitus
 GFR Glomerular filtration rate

GI Gastrointestinal

• GIP Gastric inhibitory polypeptide

GLP-1 Glucagon-like peptide 1
 GLUT4 Glucose transporter 4
 G6P Glucose-6-phosphate
 HbA1c Glycosylated hemoglobin

HbA1c Glycosylated hemoglobinHDL High-density lipoprotein

• **HHNKS** Hyperglycemic hyperosmolar nonketotic state

• HIV Human immunodeficiency virus

• **HK** Hexokinase

• **IDDM** Insulin dependent diabetes mellitus

IFG Impaired fasting glucose
 IGT Impaired glucose tolerance

• IMTG Intramyocellular triglyceride

• IRAP Insulin-responsive amino peptidase

• IR Insulin Resistance

• IRS Insulin receptor substrate

• LADA Latent autoimmune diabetes in adults

• **lb** Pound

**MET** 

LDL Low-density lipoproteinMAP Mitogen-activated protein

• **MODY** Maturity- onset diabetes of the young

Metabolic equivalent

• m RNA RNA messenger

• **NEFA** Nonesterified fatty acid

• **NIDDM** Non insulin dependent diabetes mellitus

• NLD Necrobiosis Lipoidica Diabeticorum

• **NO** Nitric oxide

• **NOS** Nitric oxide synthase

• OGTT Oral glucose tolerance test

• P protein

• PAD peripheral arterial disease

• **PDK** phosphoinositide-dependent kinase

• **PDR** Proliferative diabetic retinopathy

• PI Phosphatidylinositol I

• **PKC** Protein kinase C

PPG Postprandial plasma glucosePPHG Postprandial hyperglycemia

QOL Quality of lifeRNA Ribonucleic acid

• **SMBG** Self-monitoring of blood glucose

• **SNF-1** Sucrose non-fermenting 1

• ST Strength Training

• TSH Thyroid stimulating hormone

• VAMP-2 Vesicle-associated membrane protein-2

• VLDL Very low-density lipoprotein

• VO2 max Maximum oxygen consumption

#### **Definition of Terms**

- Acute exercise: A single bout of exercise (Musi et al, 2001).
- Blood Glucose: Sugar levels in the blood (Schwartz, 1997).

  Diabetes mellitus: Metabolic disorder of multiple etiologies characterized by chronic hyperglycemia with disturbances of carbohydrate, fat and protein metabolism resulting from defects in insulin secretion, insulin action, or both, the blood glucose level in FBG>110,in OGTT 2 hour post load glucose >200 mg/dL and in HbA1c > 6% to diagnose it as diabetes. (Tierney et al., 2002)
- Fasting Plasma Glucose (FPG): Is a blood glucose that is obtained after no caloric intake for at least eight hours. (Plasma glucose is obtained by venipuncture; whole blood glucose is most commonly obtained by fingerstick using a hand-held glucose meter. The normal range is FBG < 110 mg/dL (American Diabetes Association 2002a).
- Oral Glucose Tolerance Test(OGTT): A test to determine if a person is diabetic. The test measures the body's ability to clear sugar from the blood in a reasonable time after having taken a standardized oral dose of glucose (sugar) (American Diabetes Association 2002a).
- **HbA1C** or **A1C** (glycated hemoglobin): Reflects the mean glycemia over the preceding two to three months. Values are free of day to day glucose fluctuations and are unaffected by exercise or recent food ingestion. The interpretation of this test depends on the red blood cells having a normal life span, the average being 120 days. Persons with hemolytic disease or other conditions with a shortened red blood cell survival exhibit a significant reduction in A1C. A1C can still be used to monitor inmates with these conditions but the values must be compared with previous values from the same inmate, not from published reference values. High A1C levels have been reported in iron deficiency anemia, probably due to the high proportion of oldcirculating erythrocytes. The normal rang is HbA1c < 6%. (American Diabetes Association 2002a).
- Impaired Glucose Tolerance (IGT) and Impaired Fasting Glucose
  - (IFG): Are intermediate stages between normal glucose homeostasis and diabetes. Persons with IGT and IFG are at risk for future diabetes and cardiovascular disease. Ranged from FBG ≥ 110 mg/dL and
  - < 126 mg/dL OR OGTT 2 hour post load glucose ≥140 mg/dL and

- < 200 mg/dL (American Diabetes Association 2002a).
- Insulin: A hormone secreted by the pancreas that regulates levels of sugar in the blood (American Diabetes Association 2002a).
- Insulinopenia: Is deficient secretion of insulin by the pancreas, resulting in hyperglycemia. Called also hypoinsulinism (Rajas et al., 2000)
- Gestational Diabetes Mellitus (GDM): Is any degree of glucose intolerance identified during pregnancy (American Diabetes Association 2002a).
- Insulin Sensitivity: The body's cells are not sensitive to insulin so they resist it and sugar levels are not regulated effectively (American Diabetes Association 2002a).
- Insulin Resistance: A condition that occurs when insulin becomes ineffective or less effective than is necessary to regulate sugar levels in the blood (American Diabetes Association 2002a).

### **CHAPTER I**

## **INTRODUCTION**

The term diabetes mellitus describes a metabolic disorder of multiple etiologies characterized by chronic hyperglycemia with disturbances of carbohydrate, fat and protein metabolism resulting from defects in insulin secretion, insulin action, or both (**Tierney et al., 2002**)

Diabetes is a chronic illness that requires continuing medical care and patient self-management education to prevent acute complications and to reduce the risk of long-term complications. Diabetic care is complex and requires that many issues, beyond glycemic control, be addressed. A large body of evidence exists that supports a range of interventions to improve diabetes outcomes (McCance et al., 2004).

Type 2 diabetes mellitus is the most common form of diabetes, accounting for 90% of diabetes mellitus cases all over the world. It is a term used for individuals who have relative rather than absolute insulin deficiency. Although its management can sometimes seem straightforward, the burden of serious complications may be considerable both for the individual concerned and for the health care services in general (McIntosh, et al., 2005)

The majority of patients with this form of diabetes are obese, this obesity aggravates insulin resistance most of them have an increased percentage of body fat distributed predominantly in the abdominal region (American Diabetes Association 2007)

Maintaining good overall glucose levels is important in preventing complications of diabetes. Seriously like retinopathy and blindness would be prevented. Weight management and physical activity for people with type 2 diabetes will also improve blood glucose, blood pressure and lipid levels and thereby outcomes (**Knowler et al., 2002**).

People with this type of diabetes frequently are resistant to the action of insulin and the mechanisms of type 2 diabetes mellitus are not well known, but it was suggested that it may start with the condition called insulin resistance. Although insulin can attach normally to receptors on liver and muscle cells, certain mechanisms prevent insulin from moving glucose (blood sugar) into these cells where it can be used. Most type 2 diabetics produce variable, even normal or high, amounts of insulin. In the beginning, this amount is usually sufficient to overcome such resistance. (Harvey, 2003)

Over time, the pancreas becomes unable to produce enough insulin to overcome resistance. In type 2 diabetes mellitus, the initial effect of this stage is usually an abnormal rise in blood sugar right after a meal (called *postprandial hyperglycemia*). This effect is now believed to be particularly damaging to the body. Eventually, the cycle of elevated glucose further impairs and possibly destroys beta cells, there by stopping insulin production completely. This is made evident by *fasting hyperglycemia*, in which elevated glucose levels are present most of the time (Nayak et al., 2005).

The type 2 form of the diabetes mellitus is associated with obesity and physical inactivity. Physical activity or structured exercise training used alone or in combination with diet, insulin injections, or oral hypoglycemic drugs are the foundations of therapy for type 2 diabetes. (Snowling, et al., 2006).

Evidence for the benefit of physical activity comes from studies showing that individuals who maintain a physically active lifestyle are less likely to develop insulin resistance, impaired glucose tolerance, or type 2 diabetes. The effects of exercise training on glucose control and related physiological parameters have also been extensively studied in patients with type 2 diabetes. (**NEIL**, et al., 2006)

The effect of obesity that was statistically independent of the levels of physical activity is associated with metabolic risk factors. Moreover, physical activity displayed inverse associations with triglycerides, and fasting plasma glucose and a positive association with HDL cholesterol. Those patients spent in moderate activity more than half hour in each day had significantly less risk of high fasting glucose (**LiCl**, et al., 2006).

The beneficial effects of physical exercise on the decreased insulin sensitivity caused by detrimental lifestyle were reviewed based on experimental evidences. In epidemiological studies, disease prevention has been considered at three levels: primary (avoiding the occurrence of disease), secondary (early detection and reversal), and tertiary (prevention or delay of complications). The major purpose of physical exercise for primary prevention and treatment of lifestyle-related diseases is to improve insulin sensitivity (GIN, 2000).

It is known that, during physical exercise, glucose uptake by the working muscles rises over than basal level, depending on the intensity of the work performed. However, intense exercise provokes the release of insulin-counter regulatory hormones such as glucagon and catecholamine,