

EFFECT OF PHYSICAL EXERCISE ON INSULIN, GLUCOSE, URIC  
ACID AND LIPIDS IN DIABETES MELLITUS (D.M.).

THESIS

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# **INTRODUCTION and AIM OF THE WORK**

## INTRODUCTION AND AIM OF THE WORK

For many years, physical exercise has been considered beneficial in treatment of Diabetes mellitus. In fact, the therapeutic use of exercise has advocated at least for certain type of diabetic patients. Long term effect of physical exercise as training would be of particular importance for diabetics because of their disposition to atherosclerotic disease. (Vranic and Berger, 1979) Recent studies have been shown that glucose tolerance and homeostasis are improved by exercise programs.

Physical training increases muscle sensitivity to insulin plus better insulinization of the liver in type II diabetes mellitus. The need of exogenous insulin may be diminished. Also physical training diminishes hyperinsulinemia when insulin secretion is elevated (Pjorntrop and Krotkeiwski, 1985).

Regular exercise may decrease levels of triglycerids and low density lipoprotein and increases those of high density lipoprotein. These beneficial effects of training observed in normal subjects may extend to diabetic patients as well (Zinman, 1979). A brief periods of exercise can be effective in reducing plasma cholesterol and triglycerides levels and in increasing high density lipoprotein levels. (Bergman and Auerhahn, 1985).

The aim of this work is to study the acute effect of physical

exercise by using bicycle ergometer on insulin, glucose, lipids (Triglycerides, cholesterol, high density lipoprotein and low density lipoprotein) and uric acid in diabetes mellitus.

This is to be achieved by estimation of serum insulin, glucose, lipids (triglycerides, cholesterol, low density lipoprotein and high density lipoprotein) and uric acid before and immediately after exercise.

# Review of Literature

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## DIABETES MELLITUS

Diabetes mellitus is not a disease in the classic sense. It has no distinct and definable pathogenesis, aetiology, invariable set of clinical findings, specific laboratory tests or definition and curative therapy. Rather it should be viewed as a syndrome-a clinical entity which can involve any or all of a long list of symptoms and clinical laboratory findings which show a variable response to therapy (Porte and Halter, 1981). According to Porte and Halter (1974) diabetes mellitus has five main components consisting of : -

- a) Biochemical changes, especially with regard to carbohydrate, lipid, protein and nucleotid metabolism.
- b) Microscopic and macroscopic alteration in various organs of the body consisting especially of characteristic micro-angiopathy, precocious atherosclerosis and pancreatic islet disorder.
- c) Deficient insulin action.
- d) Increased glucagon action.
- e) Clinical manifestations due to altered hydration and osmolarity, micro-angiopathies and atherosclerosis.

**DEFINITION : -**

- In spite of its complexity, diabetes mellitus may be defined as a disorder of metabolism of which the most obvious component is a diminished ability to utilize carbohydrates, manifesting itself as hyperglycaemia, glucosuria, tendency to Ketosis and typical complication (Ghalioungui and Ghareeb, 1978).
- Diabetes mellitus (DM) is characterized in most cases by relative or absolute insulin insufficiency (Oakley, 1968) and (Feling, 1971) in which an inherited susceptibility plays an important part.
- The disease may be present for prolonged periods before any abnormality of Carbohydrate could be detected (Gerasi et al., 1967).

### CLASSIFICATION

The traditional classification of clinical diabetes mellitus is based on age of onset and recognizes juvenile onset (J.O.D.) and maturity-onset diabetes (M.O.D.) varieties.

A further presentation of diabetes in the young is described as maturity onset diabetes of young (M.O.D.Y.) (Tattersal and Fajans, 1975).

The terms "Juvenile" and "Maturity" onset are now regarded as unsatisfactory and misleading. Some patients mature in years, may develop diabetes that is insulin dependent and the presentation of a maturity onset in the young is referred to above.

The classification includes three clinical classes : -  
Diabetes mellitus, impaired glucose tolerance and gestational diabetes. In addition, the classification includes stages that may be part of the natural history of diabetes in which there are no abnormalities of Carbohydrate metabolism, namely, previous abnormality of glucose tolerance and potential abnormality of glucose tolerance (National diabetes data group, 1979).

## CLASSIFICATION

### I- Diabetes mellitus :

- a- Insulin dependent type I.
- b- Non insulin dependent type II.
  - Non obese
  - Obese

### c- Other types :

Including diabetes associated with certain condition and syndromes.

- 1- Pancreatic diseases.
- 2- Hormonal.
- 3- Drugs or chemical induced.
- 4- Insulin receptor abnormality.
- 5- Certain genetic syndromes.

### II- Impaired glucose tolerance (I.G.T.);

- Non obese
- Obese
- IGT associated with certain conditions and syndromes as:
  - Pancreatic disease.
  - Hormonal.
  - Drug or chemical induced.
  - Genetic syndromes.

III- Gestational diabetes.

IV- Previous abnormality of glucose tolerance (prev AGT).

V- Potential abnormality of glucose tolerance (Pot AGT).

Insulin Dependent Diabetes Mellitus "IDDM" or Type I .

With few exceptions diabetes mellitus in children is of the insulin dependent variety-previously called Juvenile onset diabetes mellitus "JOD" . "IDDM" however is not restricted to onset in the childhood period, but it may occur at any age (Fajan et al, 1978).

This type of diabetes mellitus is usually characterized clinically by abrupt onset of symptoms, insulinpenia and dependence on injected insulin to sustain life, and proneness of Ketosis (Rotter and Rimoin.,1978).

Anatomically : the islets are small with decreased number of Beta cells. Though hyperplasia of other islets cells that produce glucagon, somatostatin and pancreatic polypeptide is often seen (Gepts et al.,1977).

The decreased number of Beta cells in the islets results in disappearance of Beta cell function as shown by low plasma levels of the

connecting peptide molecule "C peptide" which is normally co-secreted with insulin, (Malmquist et al, 1982) and Werther et al, 1982).

Type I "IDDM" has been long associated with autoimmune diseases, such as Hashimoto's thyroiditis or Addison's disease. It is also known to be associated with an increased incidence of histocompatibility antigens, in particular HLA-B8, HLA-BW15, DW3, DW4 (Irvine et al., 1977; Rotter and Rimsin, 1978).

Subclassification of Type I Diabetes Mellitus "IDDM" :  
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Recent genetic studies, serum antibody measurements and tissue typing have provided strong evidence that type I diabetes is not individually homogeneous (Irvine 1977 ; Fajan et al 1978; Cudworth and Wolf 1982).

It was subdivided by the W.H.O. expert committee in 1980 into two groups : -

Type Ia and Type Ib.

This subdivision may aid in better prognostic information and more assured genetic counselling (Genuth, 1982 ).

Type Ia :-

This subtype occurs in young age with male preponderance. It has a genetic vulnerability to an environmental agent, thus identical

twins may not be concordant for the disease.

It has a primary association with HLA-DR3 and HLA-DR4 (Cudworth and Wolf.,1982).

The serum antibodies in this type are short term. They produce high titres of antibody to the exogenous insulin that is administered therapeutically (Genuth.,1982).

It is not usually associated with other autoimmune disturbances.

#### Type Ib :-

It occurs at middle age with a striking female preponderance (Cudworth and Wolf.,1981). In this group identical twins has as high degree of concordance for diabetes suggesting a genetically conferred inevitability of the disease. It has a strong association with HLA-DR3. Islet cell antibodies tend to persist in the serum of the patients. These patients and their families have frequently other autoimmune disorders, such as Hashimoto's disease.