

# **STUDY OF LIPOPROTEIN (a) IN OBESITY AND DIABETES MELLITUS**

## **Thesis**

Submitted for Partial Fulfillment of  
The M.Sc. Degree of  
*Endocrinology*

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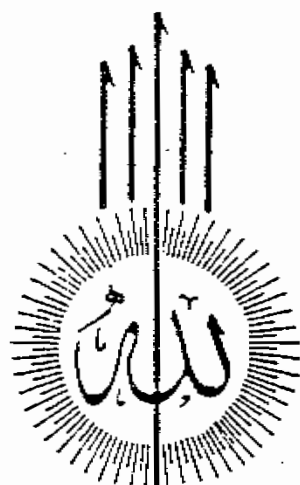
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**FACULTY OF MEDICINE  
AIN SHAMS UNIVERSITY  
1995**



قَالُوا سُبْحَانَكَ لَا عِلْمَ لَنَا إِلَّا مَا  
عَلَّمْتَنَا إِنَّكَ أَنْتَ الْعَلِيمُ الْحَكِيمُ

سَدَقَ اللَّهُ الْعَظِيمُ  
الْبَقَّة - ٣٢٢ -



To...

*My Family*

*Mohamed*

## Acknowledgment

*I express my deepest thanks to Prof. Dr. Sami Abdel Fattah, for his great help in preparing this work. Also I'm very grateful to Dr. Salah Shellbahi, for his co-operation and his great help in this work. Many thanks to Prof. Dr. Wassef Girgis, who has chosen this point for my work.*

*Also, I express my deepest thanks to Dr. Gloria, Dr. Zakaria, for doing laboratory tests in this work.*

*Lastly I'm very grateful to any one who help me in this work.*

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# **Introduction and Aim of the work**

## INTRODUCTION

Lipoprotein (a) is a special class of lipoprotein B (low density lipoprotein). The similarity of its apoprotein to plasminogen suggest a strong relationship of this lipoprotein to the process of atherogenesis.

Obese patients are more prone to develop metabolic complications such as atherosclerosis coronary heart diseases, hypertension and diabetes mellitus. These complications are more common in android obese subjects than gynoid obese subjects.

Diabetes mellitus is strong risk factor in atherogenesis but it is not known whether DM itself or through its action on lipoprotein (a) potentiates process of atherosclerosis.

### **Aim of the present work:**

To find out level of lipoprotein (a) in different types of obesity (android and gynoid).

To find out level of lipoprotein (a) in non insulin dependent diabetic patients both obese and non obese.

# **Review of Literature**

## Chapter (1)

### OBESITY

#### Definition:

Obesity is a state of abnormal accumulation of neutral fat in the storage depot of the body. It is ultimately the consequence of ingesting more calories than those needed (*Bray and Campfield, 1975*). But Hall et al. (1974) suggested that obesity is not only the adipose tissue which constitute a great portion of the total body weight but also there is increased in the lean body tissue which include extra and intracellular water and mineral mass.

Definition of obesity must take many varients into consideration which include age, sex and degree of physical activity (*Bray, 1976*).

At any moment, the individual's body weight and composition represents the cumulation balance of all previous energy intake and expenditure (*Stack and Rothwell, 1982*). Individuals may be considered overweight if they are more than 10% over the desirable weight and obese if they are more than 20% over that weight (*Ramsay, 1980*).

The most useful anthropometric measure for obesity is called body mass index (BMI) which represents weight (kg)/height (meters<sup>2</sup>). Thus according to BMI, obesity can be defined as BMI greater than 27 kg/m<sup>2</sup> for males and greater than 25 kg/m<sup>2</sup> for females (*Edwin and Jules, 1981*).

### **Classifications:**

Various classifications of obesity are present according to etiology distribution pathogenesis and onset. They include the followings:

1. Simple obesity and Secondary obesity (*Pauline, 1980*): Simple obesity is the most common and its etiology is not fully understood while secondary obesity is related to clear metabolic or endocrinal factors such as hypothyroidism, hypogonadism and polycystic ovary syndrome. Incidence of secondary obesity is less than 1% (*Bireman, 1985*).
2. Hypercellular and Normocellular obesity: Hirsch and Knittle 1970 classified obese persons into normocellular if they have the normal number of fat cells and hypercellular if the number of fat cells is increased, both types are hypertrophic as they have enlarged adipocytes. Most studies found that weight reduction by diet and exercises is

due to decrease in fat cell size but not in fat cell number (Olefsky, 1987).

3. Android and Gynoid obesity: Vague et al. (1985) classified obesity according to anatomical distribution of adipose tissue into android obesity or upper segment obesity (fat mainly deposited in abdomen) and gynoid obesity or lower segment obesity (fat deposited in buttocks and thighs). Vague (1985) and Bjorntrap (1987) found higher incidence of complications as diabetes mellitus, coronary heart diseases, hypertension and embolism in android obesity.
4. Life long obesity and Adult onset obesity: Joseph et al. (1983) classified obesity according to time of onset into life long obesity that begins in childhood and adult onset obesity which has a middle age onset.
5. Constitutional, Reactive and Developmental: Bruch (1973) divided obesity into 3 main types, constitutional obesity due to genetic or physiological causes. Reactive obesity due to overeating in response to emotional distress and developmental obesity as reactive but occur in childhood.

6. Visceral and Subcutaneous obesity: obesity is classified according to the site of depot fat in the abdomen whether in abdominal cavity (visceral) or in the subcutaneous tissue (subcutaneous) as demonstrated by CT (*Fujioka et al., 1987*). Visceral obesity is associated with metabolic complications as insulin resistance, hyperlipidemia and glucose intolerance (*Kissebah et al., 1982*).

#### **Factors prediposing to obesity:**

1. Decline in physical activity: physical activity normally declines during adult life resulting in decreased energy expenditure if caloric intake remains unchanged, there will be gain of weight (*Bjorntrop et al., 1977*). Once obesity is established physical activity is restricted (*Curtis and Brandfield, 1971*) (*Durnin, 1984*) but the reduced level of activity may be partly balanced by increased energy cost of weight supported activity (*Blair and Buskirk, 1987*). Decline in physical activity together with high fat diet is essential for development of massive obesity (*Godeau and Krempf, 1993*).
2. Diet: in order to discuss the role of diet in development of obesity the following points should be fulfilled: Fats stored in the body comes mainly from dietary fat as they are more