

بسم الله الرحمن الرحيم



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شبكة المعلومات الجامعية التوثيق الالكتروني والميكرو فيلم



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جامعة عين شمس

التوثيق الإلكتروني والميكروفيلم

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بالرسالة صفحات

لم ترد بالأصل



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SERUM LEPTIN IN INSULIN DEPENDENT DIABETES MELLITUS

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THESIS

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Introduction

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Introduction

Leptin, the product of the *ob* gene, is recently discovered proteohormone with a molecular weight of 16 KD a that is thought to play a key role in the regulation of body weight. Although its amino acid sequence exhibits no major homologies with other proteins, prediction of its tertiary structure shows similarities with class I cytokines (Rock et al., 1996). This is not surprising in that leptin receptors belong to the cytokine class I receptor family. Leptin receptors are found ubiquitously in the body indicating a general role (Chen et al., 1996). Leptin is produced by differentiated adipocytes (Rentsch et al., 1996). It acts on the hypothalamus, thereby suppressing food intake and stimulating energy expenditure (Levin et al., 1996). Besides its metabolic effects, leptin affects a number of endocrine axes. In this regard a role for leptin in the control of pulsatile growth hormone, ACTH, thyroxin and lutinising hormone secretion has been demonstrated (Werner et al., 1997).

The most important variable that determines circulating leptin levels is body fat mass. Obviously, under condition of regular eating cycles, leptin reflects the proportion of the adipose tissue (Kolaczynski et al 1996).

This constitutive synthesis of leptin is modulated by a number of hormones. Strong stimulators in rodents and humans are insulin and glucocorticoids (Kiess et al., 1996). Growth hormone excrets short-term a stimulating and long-term suppressing effect (Blum WF et al., 1997).

In order to understand the behavior of leptin in children with IDDM, it is essential to analyze the main data concerning the relationship between leptin & insulin. In fact, in IDDM there is

insulin deficiency and in some pathological conditions this insulin deficiency is associated with insulin resistance (Kamoda et al., 1998- Banerji et al., 1999).

It is now well known that leptin is an important factor which can modify serum insulin levels and antagonize insulin signaling (probably by decreasing insulin-induced tyrosin phosphorylation of insulin receptor substrate-1). In human, however, reports on the influence of insulin on leptin are conflicting (Taylor SI et al 1996 – Segal et al., 1996).

The major determination of serum leptin levels in IDDM patients are BMI, sex, pubertal stage and independent of pubertal stage; age (Hickey MS, et al., 1996). It has been demonstrated that the serum levels of leptin were significantly higher in obese non diabetic patients and positively correlated with BMI (Considine Rv et al., 1997-Pi-Sunyer et al., 1999). These finding suggest that obese humans may be resistant to the effects of leptin in modulating food intake and energy expenditure (Considine RV et al., 1996).



Aim Of The Work

AIM OF THE WORK

The aim of this work is to study the role of leptin in IDDM and the relationship between serum levels of leptin and insulin in obese and non obese children.



Review Of Literature

REVIEW OF LITERATURE

Diabetes mellitus

Definition

Clinical diabetes mellitus (DM) is a syndrome of metabolic disease characterized by hyperglycemia due to either an absolute deficiency of insulin secretion or a reduction in the biological effectiveness of insulin and results in abnormal metabolism of carbohydrates ,proteins & fats (Green et al 1994) .

Insulin dependent DM is the most common childhood endocrine metabolic disorder with important consequences for physical and emotional development.

Etiological classification of diabetes mellitus:

A. Type I insulin dependent diabetes mellitus

This type is characterized by severe insulinopenia and dependence on exogenous insulin to prevent ketosis and to preserve life.

B. Type II non insulin dependant diabetes mellitus

It may range from predominantly insulin resistance with relative insulin deficiency to a predominantly secretory defect with insulin resistance.

C. Other specific types e.g. gestational diabetes, endocrine diseases (hyperthyroidism , Cushing diseases), (Mandrup-Poulsen et al., 1998). ect. Table (a)