



EFFECT OF ADIPONECTIN LEVEL IN TYPE II DIABETIC POSTMENOPAUSAL WOMEN COMPARED TO HEALTHY WOMEN

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ABSTRACT

A study was conducted to investigate the regulatory roles of adiponectin concentration in diabetic type II postmenopausal women and to study the correlation between the parameters (serum glucose, cholesterol and triglyceride). A total of sixty subjects involved in this study; thirty diabetic women (BMI<30) and thirty healthy postmenopausal women (BMI<30), selected from Baghdad teaching hospital in Baghdad/ Iraq. ELISA (enzyme linked immune sorbent assay) technique was used for the measurement of serum adiponectin. Blood glucose, cholesterol and triglyceride were determined by using colorimetric method. Data was expressed as mean \pm SD (standard deviation). Means were compared by student t-test. Results revealed that the level of serum adiponectin in diabetic postmenopausal women were significantly ($P < 0.01$) lower than that of non diabetic postmenopausal women. On the other hand, serum glucose in diabetic postmenopausal women was significantly ($P < 0.01$) higher than non-diabetic women. The present study indicates the possibility of future development of new anti-diabetic agents that act independent of insulin action.

KEY WORDS: Adiponectin, diabetes mellitus, postmenopausal women.

INTRODUCTION

Adiponectin is a protein hormone (244-amino-acid-long polypeptide) (Shapiro and Scherer, 1998) that modulates a number of metabolic processes, including glucose regulation and fatty acid oxidation (Díez and Iglesias, 2003) Adiponectin is exclusively secreted from adipose tissue (and also from the placenta in pregnancy) into the bloodstream and is very abundant in plasma relative to many hormones (Chen *et al.*, 2006). Levels of the hormone are inversely correlated with body fat percentage in adults (Ukkola and Santaniemi, 2002). Coppola (Coppola *et al.*, 2008) reported that adiponectin is secreted into the bloodstream where it accounts for approximately 0.01% of all plasma protein at around 5-10 μ g/mL.

Adiponectin automatically self-associates into larger structures. Initially, three adiponectin molecules bind together to form a homotrimer. The trimers continue to self-associate and form hexamers or dodecamers. Like the plasma concentration, the relative levels of the higher-order structures are sexually dimorphic, where females have increased proportions of the high-molecular weight forms. Recent studies attributed the high-molecular weight to the biological active form regarding glucose homeostasis (Oh *et al.*, 2007). The human homologue was identified as the most abundant transcript in adipose tissue. Contrary to expectations, despite being produced in adipose tissue, adiponectin was found to be decreased in obesity (Ukkola and Santaniemi, 2002; Díez and Iglesias, 2003; Nedvídková *et al.*, 2005). This down regulation has not been fully explained. The gene was localized to chromosome 3q27, a region highlighted as affecting genetic susceptibility to type 2 diabetes and obesity. Supplementation by differing forms of adiponectin was

able to improve insulin control, blood glucose and triglyceride levels in mouse models (Nedvídková *et al.*, 2005).

MATERIAL & METHODS

A sample of thirty female's patients with type II diabetic mean age (57 ± 3.4) range (49-68 years) and thirty healthy females mean age (59 ± 4.0) range (55-68 years) included in this study. The subjects were selected from the people attending the out-patient clinic in medical city-Baghdad teaching hospital during the period between October and January 2013. Enzyme linked immune sorbent assay (ELIZA) was used for the measurement of serum adiponectin concentration. Colorimetric method was used in the determination of serum cholesterol, triglyceride and serum glucose. The weight and height were used to calculate body mass index (BMI), data expressed as mean \pm SD results.

Statistical analysis was performed using SPSS-21 (Statistical Packages for Social Sciences- version 21). Student T-test was used to assess significant difference between means.

RESULTS

Table (1) illustrates that the level of serum adiponectin in diabetic women is significantly ($P < 0.01$) lower than non-diabetic women; whereas serum glucose level in diabetic women is significantly ($P < 0.01$) higher than non-diabetic women. On the other hand, no significant differences were detected between means of BMI (body mass index), total cholesterol and triglyceride in diabetic and non-diabetic women.

TABLE 1: Means \pm SD for non-diabetic and diabetic type II postmenopausal women

parameters	Type II diabetic postmenopausal women	Non-diabetic postmenopausal women
No. of observations	30	30
BMI (kg/m)	26 \pm 3.6	25 \pm 4.4
Serum adiponectin (μ g/ml)	4.8 \pm 1.3	9.4 \pm 2.8
Serum cholesterol (mg/dl)	153 \pm 49.3	157 \pm 26.7
Serum triglyceride (mg/dl)	107 \pm 20.6	117 \pm 31.3
Serum glucose (mg/dl)	175 \pm 36	88 \pm 12.6

** (P < 0.01)

DISCUSSION

Many evidences from animal and human studies show that adiponectin plays an important role in the pathophysiology of insulin resistance, lipid metabolism (Matsubara *et al.*, 2002) diabetes (Kubota *et al.*, 2002; Lindsay *et al.*, 2002), and inflammation (Yokota *et al.*, 2000). These will increase risk for cardiovascular disease (Yokota *et al.*, 2000). The result of present study shows that the level of serum adiponectin in diabetic women is significantly lower than non-diabetic women. Similar result was reported by Coppola *et al.* (2008) and Araki *et al.* (2009) who demonstrated that the levels of adiponectin are reduced in diabetics compared to non-diabetics. Weight reduction significantly increases circulating levels (Coppola *et al.*, 2008).

Human adiponectin has 244 amino acids, and the molecular weight of the monomer is 26,413 (Scherer *et al.*, 1995). However, it circulates in polymeric form. Plasma concentrations reveal a sexual dimorphism, so females having higher levels than males. Circulating adiponectin levels and adiponectin gene expression in adipose tissues are also found to be lower in such patients (Hug *et al.*, 2004; Fang and Sweeney, 2006). Hypoadiponectinemia has been implicated in increased risk of coronary artery disease (Fang and Sweeney, 2006).

High-molecular-weight adiponectin was further found to be associated with a lower risk of diabetes with similar magnitude of association as total adiponectin (Zhu *et al.*, 2010). Adiponectin appears to be linked to glucose homeostasis since plasma adiponectin levels are lower in diabetic subjects (Hotta *et al.*, 2000; Haque *et al.*, 2002) and are positively correlated with glucose utilization (Weyer *et al.*, 2001).

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