

**ISSN: 2308-6513****E-ISSN: 2310-3434****Founder: Academic Publishing House Researcher****DOI: 10.13187/issn.2308-6513**

Has been issued since 2013.

**European Journal of Medicine**

UDC 61

**Effect of Adiponectin Level in Type II Diabetic Postmenopausal Women Compared to Healthy Women**<sup>1</sup>Mohamed S. Al-Braich<sup>2</sup>Nada K. Al-Husaini<sup>3</sup>Sama H. M. Saleh<sup>4</sup>Munira F. Awn<sup>1</sup>University of Baghdad, Iraq

Assist. Prof. / section of Biochemistry, College of Dentistry

<sup>2</sup>University of Baghdad, Iraq

Lecturer / section of Biochemistry, College of Dentistry

<sup>3</sup>Institute of Medical Technology, Foundation of Technical Education, Iraq

Assist. lecturer in Anesthesia department

<sup>4</sup>University of Baghdad, Iraq

Lecturer / section of Histology, College of Dentistry

**Abstract.** Adiponectin is protein secreted from adipose tissue and plays important role in regulating glucose level as well as fatty acid breakdown. Adipose tissue has been considered an important endocrine organ. Adiponectin has been shown to completely reverse insulin resistance. A total of sixty subjects involved in this study to examine 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). Thirty of each diabetic women (BMI<30) and healthy postmenopausal women (BMI<30) were 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. Results showed that the level of serum adiponectin in diabetic postmenopausal women were significantly ( $P < 0.01$ ) lower than that of non diabetic postmenopausal women. 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.

**Keywords:** Adiponectin; diabetes mellitus; postmenopausal women.

**Introduction.**

Adiponectin is a protein hormone (244-amino-acid-long polypeptide) <sup>(1)</sup> that modulates a number of metabolic processes, including glucose regulation and fatty acid oxidation <sup>(2)</sup>. 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 <sup>(3)</sup>. Levels of the hormone are inversely correlated with body fat percentage in adults <sup>(4)</sup>.

Coppola<sup>(5)</sup> reported that adiponectin is secreted into the bloodstream where it accounts for approximately 0.01% of all plasma protein at around 5-10 µg/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 <sup>(6)</sup>.

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 <sup>(2, 4, 7)</sup>. 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 <sup>(7)</sup>.

### Material and methods.

A sample of thirty female's patients with type II diabetic mean age ( $57 \pm 3.4$ ) range (49-68 years) and thirty healthy females mean age ( $59 \pm 4.0$ ) 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.

The characteristics of non-diabetic and diabetic type II postmenopausal women are shown in table 1.

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 investigation	30	30
BMI (kg/m)	$26 \pm 3.6$	$25 \pm 4.4$
Serum adiponectin ( $\mu\text{g/ml}$ )	$4.8 \pm 1.3$	$9.4 \pm 2.8^{**}$
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)	$175 \pm 36$	$88 \pm 12.6^{**}$

This table 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.

### Discussion

Many evidences from animal and human studies show that adiponectin plays an important role in the pathophysiology of insulin resistance, lipid metabolism<sup>(8)</sup>, diabetes <sup>(9, 10)</sup> and inflammation<sup>(11)</sup>. These will increase risk for cardiovascular disease<sup>(11)</sup>. 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<sup>(5)</sup> and Araki<sup>(12)</sup> who demonstrated that the levels of adiponectin are reduced in diabetics compared to non-diabetics. Weight reduction significantly increases circulating levels<sup>(5)</sup>.

Human adiponectin has 244 amino acids, and the molecular weight of the monomer is 26,413 <sup>(13)</sup>. 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 <sup>(14, 15)</sup>. Hypoadiponectinemia have been implicated in increased risk of coronary artery disease <sup>(15)</sup>.

High-molecular-weight adiponectin was further found to be associated with a lower risk of diabetes with similar magnitude of association as total adiponectin. <sup>(16)</sup> Adiponectin appears to be linked to glucose homeostasis since plasma adiponectin levels are lower in diabetic subjects <sup>(17, 18)</sup> and are positively correlated with glucose utilization <sup>(19)</sup>.

### References:

1. Shapiro L, Scherer PE (1998). "The crystal structure of a complement-1q family protein suggests an evolutionary link to tumor necrosis factor". *Curr. Biol.*8 (6): 335–8.
2. Díez JJ, Iglesias P (2003). "The role of the novel adipocyte-derived hormone adiponectin in human disease". *Eur. J. Endocrinol.*148 (3): 293–300.
3. Chen J, et al. (2006). "Secretion of adiponectin by human placenta: differential modulation of adiponectin and its receptors by cytokines.". *Diabetologia*49 (6): 1292–302.
4. Ukkola O, Santaniemi M (2002). "Adiponectin: a link between excess adiposity and associated comorbidities?". *J. Mol. Med.*80 (11): 696–702.
5. Coppola A, Marfella R, Coppola L, Tagliamonte E, Fontana D, Liguori E, Cirillo T, Cafiero M, Natale S, Astarita C (2008). "Effect of weight loss on coronary circulation and adiponectin levels in obese women". *Int. J. Cardiol.*134 (3): 414–6.
6. Oh DK, Ciaraldi T, Henry RR. (2007). Adiponectin in health and disease. *Diabetes Obes Metab.*9:282–289.
7. Nedvídková J, Smitka K, Kopský V, Hainer V (2005). "Adiponectin, an adipocyte-derived protein". *Physiol Res*54 (2): 133–40.
8. Matsubara M, Maruoka S, Katayose S (2002). Inverse relationship between plasma adiponectin and leptin concentrations in normal-weight and obese women. *Eur J Endocrinol* 147:173–180.
9. Lindsay RS, Funahashi T, Hanson RL, Matsuzawa Y, Tanaka S, Tataranni PA, Knowler WC, Krakoff J (2002). Adiponectin and development of type 2 diabetes in the Pima Indian population. *Lancet* 360:57–58.
10. Kubota N, Terauchi Y, Yamauchi T, Kubota T, Moroi M, Matsui J, Eto K, Yamashita T, Kamon J, Satoh H, Yano W, Froguel P, Nagai R, Kimura S, Kadowaki T, Noda T (2002). Disruption of adiponectin causes insulin resistance and atherosclerosis in mice. *J Biol Chem* 277:25863–25866.
11. Yokota T, Oritani K, Takahashi I, Ishikawa J, Matsuyama A, Ouchi N, Kihara S, Funahashi T, Tenner AJ, Tomiyama Y, Matsuzawa Y (2000). Adiponectin, a new member of the family of soluble defense collagens, negatively regulates the growth of myelomonocytic progenitors and the functions of macrophages. *Blood* 96:1723–1732.
12. Araki T., Emoto M., Konishi T., Ikuno Y., Lee E., Teramura M (2009). Increases high density lipoprotein cholesterol via increasing adiponectin levels in type 2 diabetes mellitus. *Metabolism Clinical and Experimental*, 58: 143-148.
13. Scherer PE, Williams S, Fogliano M, Baldini G, Lodish HF (1995). A novel serum protein similar to Clq, produced exclusively in adipocytes. *J Biol Chem* 270: 26746–26749.
14. Hug C, Wang J, Ahmad NS, Bogan JS, Tsao TS, Lodish HF (2004). "T-cadherin is a receptor for hexameric and high-molecular-weight forms of Acrp30/adiponectin". *Proc Natl Acad Sci U S A*101 (28): 10308–13.
15. Fang X, Sweeney G (2006). "Mechanisms regulating energy metabolism by adiponectin in obesity and diabetes". *Biochem. Soc. Trans.*34 (Pt 5): 798–801.
16. Zhu N, Pankow JS, Ballantyne CM, Couper D, Hoogeveen RC, Pereira M, Duncan BB, Schmidt MI (2010). "High-molecular-weight adiponectin and the risk of type 2 diabetes in the ARIC study". *J. Clin. Endocrinol. Metab.*95 (11): 5097–104.
17. Hotta K, Funahashi T, Arita Y, Takahashi M, Matsuda M, Okamoto Y, Iwahashi H, Kuriyama H, Ouchi N, Maeda K, Nishida M, Kihara S, Sakai N, Nakajima T, Hasegawa K, Muraguchi M, Ohmoto Y, Nakamura T, Yamashita S, Hanafusa T, Matsuzawa Y (2000). Plasma concentrations of a novel, adipose-specific protein, adiponectin, in type 2 diabetic patients. *Arterioscler Thromb Vasc Biol* 20: 1595–1599.

18. Haque W, Shimomura I, Matsuzawa Y, Garg A (2002). Serum adiponectin and leptin levels in patients with lipodystrophies. *J Clin Endocrinol Metab* 87: 2395–2398.
19. Weyer C, Funahashi T, Tanaka S, Hotta K, Matsuzawa Y, Pratley RE, Tataranni PA (2001). Hypoadiponectinemia in obesity and type 2 diabetes: close association with insulin resistance and hyperinsulinemia. *J Clin Endocrinol Metab* 86: 1930–1935.