





Research Article:

## Adiponectin and Total Antioxidant Capacity and Their Association with Insulin Resistance Among Women with Polycystic Ovarian Syndrome

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### Abstract

**Background and objectives:** Polycystic ovary syndrome (PCOS) is a hormonal disorder characterized by an imbalance in women's reproductive hormones within the endocrine system. PCOS is related to metabolic and endocrine disorders, including insulin resistance, glucose intolerance, obesity, diabetes mellitus, acne, and hirsutism. Obesity is common in PCOS-affected women, and it leads to abnormalities in adipocyte function and adipokine levels, including adiponectin. Adiponectin is a homeostatic regulatory agent for glucose, lipids, and insulin by its anti-inflammatory, antioxidant, and anti-fibrotic actions. It is hypothesized that the metabolic and endocrine disturbances observed in PCOS women may be related to altered adiponectin levels. **Methods:** Thirty-two women with PCOS under 35 years-old were enrolled in the current study, and 32 healthy women were matched for age with the previous group to evaluate adiponectin levels, total antioxidant capacity, glucose, and insulin resistance. **Results:** Compared with the control group, women with PCOS had significantly reduced levels of total antioxidant capacity, and adiponectin ( $P < 0.05$ ), with a concomitant significant increase in glucose and insulin levels and insulin resistance ( $P < 0.0001$ ). Interestingly, no statistically significant correlations were observed between the parameters. **Conclusions:** Accordingly, we concluded that lower adiponectin levels and total antioxidant capacity together with higher glucose and insulin levels and insulin resistance may offer predictive risk factors for PCOS in women who are obese with fewer PCOS symptoms.

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## 1. Introduction

Polycystic ovarian syndrome (PCOS) is one of the most prevalent metabolic-endocrine diseases that affects females who are of reproductive age. (1). Genetic factors have a major role in the development of PCOS. The disease has a variable penetrance that is impacted by genetic and environmental factors (2). Either the adrenal and/or ovarian is a cause of the

hyperandrogenism (3). According to molecular and genetic research, theca cells from polycystic ovaries have an intrinsic steroidogenic defect that increase androgen production (4). Consequently, this results in increased levels of three enzymes in the steroidogenic pathway: side chain cleavage enzyme, 3- $\beta$  hydroxyl steroid dehydrogenase, and 17- $\alpha$  hydroxyls. Adiponectin (APN) is a necessary protein that is synthesized and secreted by adipose tissue. Adipose tissue has long been believed to be an inert organ used for fat storage (5). Interestingly, adipokines, a class of physiologically active substances released by adipose tissue both locally and systemically, are increasingly recognized as metabolically active substances that contribute to a range of metabolic activities. Adiponectin (APN) also has additional benefits, including atheroprotective, anti-apoptotic, renoprotective, cardioprotective, vasoprotective, antioxidant, and anti-inflammatory (6).

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The pathophysiology of PCOS has recently been connected to adipocytokines, specifically APN. In healthy individuals, adiponectin levels were found to be inversely linked with obesity and to be controlled by the degree of insulin resistance and hyperinsulinemia. Studies showed that females with PCOS have lower levels of circulating APN, which exacerbates PCOS-related problems (7). The increased obesity or insulin resistance that are commonly observed in these women may have caused the altered levels, or they may have developed spontaneously. A vicious cycle may arise from the lower APN levels because of the subsequent reduction in insulin sensitivity and insulin resistance (8). Obesity is common in PCOS-affected women, and it leads to abnormalities in APN. It is supposed that the metabolic and endocrine abnormalities observed in women with PCOS may potentially be related to variations in APN levels. APN levels may be restored by treatment interventions and weight loss. Evaluating the levels of APN in women with PCOS may help to clarify the pathogenesis and consequences of the disease. According to some findings, women with PCOS had considerably higher serum APN levels, whereas other studies discovered that PCOS patients had lower serum APN levels (9).

Antioxidants were evaluated because they may help manage PCOS by lowering oxidative stress and alleviating symptoms (10). Because they are potent antioxidants, vitamins C and E shield cells from oxidative damage, which aids in the neutralization of reactive oxygen species (11). Total antioxidant capacity (TAC) can be defined as the capacity of serum to decrease the formation of free radicals, in addition to protect cell structures from the harmful effects of these radicals (12). These can be regarded as one of the antioxidant fortifications in the body. TAC can be disrupted by any change in the plasma level of antioxidant and/or oxidative stress. TAC can be performed as a diagnostic and progress test in the workup of PCOS (13). The total antioxidant content of a patient's serum is measured by TAC. Moreover, TAC in follicular fluid produces a microenvironment for the developing oocyte and directly affects oocyte quality, which rises as follicles enlarge due to TAC's developmental capacity. These roles might be undermined by insufficient antioxidant in follicular fluid (14).

The current study aimed to evaluate the serum APN and total antioxidant capacity levels in women with PCOS, and its correlation with glucose and insulin resistance.

## 2. Material and Methods

### 2.1. Patients

Sixty-four women were included in the current case-control study, 32 healthy women under 35 years old as the control group, and 32 women with PCOS were matched for age with the control group. These women were diagnosed with PCOS using the Rotterdam criteria (15). The Rotterdam criteria states that oligo-anovulation, hyperandrogenism, and polycystic ovaries (defined as having at least 12 follicles measuring 2–9 mm in diameter and/or an ovarian volume > 10 mL in at least one ovary) are the three criteria that define PCOS. After providing their informed consent, all participants were included.

### 2.2. Exclusion criteria

We excluded any conditions or medications that affect total antioxidant or adiponectin levels. Additionally, women on steroids, oral contraceptives, thiazolidinediones, metformin, or hormone therapy were not included in the current study.

### 2.3. Laboratory analysis

Five mL of fasting venous blood samples were collected from the healthy individuals and patients into tubes. After 10 min. incubation in a water bath, serum was separated by centrifugation at 3,500 rpm for 10 min. Except for glucose levels, serum samples were stored at -20°C until estimation of adiponectin, TAC, and insulin levels.

The enzymatic colorimetric technique was utilized to determine the FSG, and a kit by BIOLABO (France) was provided. Serum insulin was measured using an enzyme-linked immunosorbent test equipment with a kit from Monobind kit (USA).

### 2.4. Methods

Enzyme-Linked Immuno Sorbent Assay was used to determine the serum levels of adiponectin, insulin (measured at 450 nm), and total antioxidant capacity (measured at 520 nm) by kits supplied by USBIOLOGICAL (USA), Monobind (USA), and Human Total antioxidant status (TAC), respectively (16). The minimum detectable doses for these kits are typically less than 0.68 ng/ml. for USBIOLOGICAL (USA), HRP 1 x 10<sup>-18</sup> moles ALP 1x10<sup>-21</sup> moles for Monobind (USA), and 0.23 mmol Trolox Equiv./L for Human Total antioxidant status (TAC). Glucose levels were determined by enzymatic colorimetric method, utilizing a kit provided from BIOLABO (France). Serum glucose and insulin levels were utilized to determine insulin resistance (IR) by the following equation:  $HOMA-IR = \text{Serum Insulin levels} \times \text{Serum Glucose levels} / 22.5$  (17).

### 2.5. Statistical analysis

Data were represented as mean ± standard deviation (SD). Unpaired t-tests were utilized to compare the control group and women with PCOS, using GraphPad Prism (10.0), USA. Statistical significance was represented at  $p < 0.05$ .

## 3. Results

The body mass index (BMI) and age of the healthy and PCOS participants are represented in Table 1. Sixty-four females enrolled in the current study, thirty-two healthy women utilized as a control group and thirty-two women with PCOS. There was no significant variation in the age between healthy (25.10 ± 6.656) and PCOS women (24.83 ± 6.481) ( $p$ -value 0.8). Nevertheless, a significant variation in the BMI was observed between the healthy and PCOS women ( $p < 0.0001$ ). Females with PCOS had significantly higher body mass index than healthy control (difference between means 9.185 ± 1.478) **Table 1**.

**Table 1.** Baseline characteristics of the healthy and PCOS participants

Parameter	Control (n=32)	PCOS (n=32)
Age (years)	25.10 ± 6.656	24.83 ± 6.481
BMI (kg/m <sup>2</sup> )	24.75 ± 7.333	33.93 ± 3.431****

Data were set as mean ± SD. Unpaired t-test was utilized, with \*\*\*\* indicating significant variations at p-value < 0.0001.

Adiponectin, total antioxidant capacity, glucose, insulin, and insulin resistance were shown in **Table 2**. PCOS women exhibited significantly lower levels of adiponectin and total

antioxidant capacity, along with significantly higher levels of glucose, insulin, and insulin resistance compared to healthy control.

**Table 2.** Serum levels of adiponectin, total antioxidant capacity, glucose, insulin, and Homeostatic model assessment for insulin resistance (HOMA-IR) in the study groups

Parameter	Control (n=32)	PCOS (n=32)	P-value
Adiponectin (µg/ml)	10.20 ± 1.697	9.246 ± 1.826*	< 0.05
TAC (U/mL)	14.01 ± 1.345	13.31 ± 1.097*	< 0.05
FSG (mg/dl)	96.43 ± 11.45	128.9 ± 36.91****	< 0.0001
Insulin (µu/L)	7.328 ± 2.564	16.02 ± 3.380****	< 0.0001
HOMA-IR	1.752 ± 0.7033	5.070 ± 2.046****	< 0.0001

Data were set as mean ± SD.

An unpaired t-test was utilized, in which \*p-value < 0.05; \*\*\*\*p-value < 0.0001 represents statistically significant variations.

#### 4. Discussion

In the present study, serum adiponectin levels were significantly lower in women with PCOS than in the control group. Several studies have also evaluated the relationship between adiponectin and PCOS (18, 19). Many findings showed that adiponectin levels in PCOS women are significantly lower than in healthy control (19, 20). In the current study,

Table 1 showed that PCOS women were obese compared to control group. Various theories behind the lower levels of adiponectin in PCOS women. Some findings revealed that adiponectin levels are unaffected by insulin resistance and fluctuates with the degree of obesity, while other findings indicate that variations in adiponectin levels are caused by glucose intolerance and insulin resistance (21). Lean or obese women with PCOS were hypoadiponectinaemia with various degrees of insulin resistance (22). Nevertheless, a previous study revealed that the lower levels of total and high molecular weight (HMW) adiponectin in PCOS-affected women occurred independently of insulin resistance and body mass index and that posttranscriptional/translational variations are behind the low levels of high molecular weight adiponectin in women with PCOS (23). Dunaif (1997) revealed that PCOS-affected women had significantly decreased expression of adiponectin mRNA in the adipose tissue when compared to BMI-matched women without PCOS (24). It observed that the lower expression of adiponectin mRNA in both subcutaneous and visceral fat tissue is consistent with the lower serum levels of adiponectin seen in women with PCOS (18). An analysis of the adipose tissue in women with PCOS suggests that the increased adiposity, as revealed by a higher body mass index in the current study, may be the reason for the lower levels of adiponectin in these patients.

Compared to controls, non-obese and young women with PCOS had higher levels of adiponectin, according to Arikan et al. (25). Because it plays a role in carbohydrate and lipid metabolism, adiponectin actively participates in energy homeostasis. It has been shown that adiponectin regulates the enzymes in charge of lipid metabolism as well as the lipoproteins high in triglycerides (26).

TAC, an antioxidant biomarker that assesses bodily fluids' capacity for antioxidants, is markedly reduced in several disorders (27, 28). In the current study, women with PCOS had significantly lower TAC levels compared to healthy controls. These findings are generally in agreement with earlier studies that found PCOS women had lower TAC levels than healthy control (29, 30). In women with PCOS, TAC can be utilized to prevent the development of insulin resistance (HOMA-IR) and oxidative stress by regular evaluations (26). Additionally, it can be used as an indicator in early diagnosis of PCOS (31-33).

In the current study, no significant correlations have been found in the general characteristics between study groups. Conflicting results concerning correlations, some studies showed that age negatively correlates with TAC (34), whereas other findings showed that the BMI have been decreased with TAC levels (35, 36). Other studies showed that BMI and age had no effects on TAC levels and variations in these characteristics were not significant in the study groups. Other studies evaluated the effects of basic characteristics on TAC levels and revealed that TAC levels decreased with increasing age (37). Accordingly, the mean age values in the study groups in our study were close, which does not affect our findings. Moreover, the present study showed that as the BMI increased, TAC levels decreased in underweight women. However, these variations were not significant. Many studies reported a negative correlations between BMI and TAC levels (34, 37). Solmi et al. also showed that underweight women had higher oxidative stress levels than the control group (38). Furthermore, Risi et al. reported in a narrative review that the underweight and obesity are two sides of one coin (39).

Several studies evaluated the relationship between glucose, IR, and PCOS (40, 41). Women with PCOS are more likely to develop IR, which has a substantial detrimental effect on health and is associated with a high incidence of IR in insulin-responsive tissues as well as aberrant insulin signalling (40). In this study, IR and glucose levels were significantly higher in women with PCOS compared to healthy control. Although the exact cause of insulin resistance syndrome (IR) in PCOS is unknown, it is thought to involve genetic and epigenetic modifications, inflammation, obesity, hyperandrogenaemia, and impaired insulin signal transduction (40). IR may specifically impact the metabolic or mitotic processes in a variety of PCOS organs, including the ovaries (42). In order to increase IR in PCOS patients, effective preventative and therapy strategies should be considered. In addition to boosting ovulation and lowering testosterone levels, lifestyle changes and insulin sensitization treatment might be useful methods for enhancing insulin sensitivity (40).

### 5. Limitations

Large sample sizes and follow-up studies are necessary for the assessment of serum adiponectin levels in PCOS women, as our study did not have sufficient sample size or follow-up.

### 6. Conclusion

Serum levels of adiponectin are lower in obese women with PCOS with concomitant higher levels of glucose and insulin resistance. These findings imply that obesity-related dietary status may exacerbate insulin resistance in the pathophysiology of PCOS by lowering blood levels of adiponectin. Accordingly, by reducing adiponectin serum level, together with nutritional status of obesity, might contribute to insulin resistance in the pathogenesis of PCOS.

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#### الأديبونيكتين والقدرة الكلية لمضادات الأكسدة وارتباطهما بمقاومة الأنسولين لدى النساء المصابات بمتلازمة المبيض المتعدد الكيسات

#### الخلاصة

الخلفية والأهداف: متلازمة المبيض المتعدد الكيسات (PCOS) هي اضطراب هرموني يتميز بخلل في الهرمونات التناسلية للمرأة داخل نظام الغدد الصماء. ترتبط متلازمة تكيس المبايض باضطرابات التمثيل الغذائي والغدد الصماء، بما في ذلك مقاومة الأنسولين، وعدم تحمل الجلوكوز، والسمنة، ومرض السكري، وحب الشباب، والشعرانية. السمنة شائعة عند النساء المصابات بمتلازمة تكيس المبايض، وتؤدي إلى خلل في وظيفة الخلايا الشحمية ومستويات الأديبونيكتين، بما في ذلك الأديبونيكتين. الأديبونيكتين هو عامل تنظيمي مهم للجوكوز والدهون والأنسولين من خلال تأثيراته المضادة للالتهابات ومضادات الأكسدة والمضادة للتليف. من المفترض أن الاضطرابات الأيضية والغدد الصماء التي لوحظت لدى النساء المصابات بمتلازمة تكيس المبايض قد تكون مرتبطة بتغير مستويات الأديبونيكتين. **الطرق:** تم تسجيل 32 امرأة مصابة بمتلازمة تكيس المبايض تحت سن 35 عامًا في الدراسة الحالية، وتمت مطابقة عمر 32 امرأة سليمة مع المجموعة السابقة لتقييم مستويات الأديبونيكتين، والقدرة الكلية لمضادات الأكسدة، والجلوكوز، ومقاومة الأنسولين. **النتائج:** بالمقارنة مع المجموعة الضابطة، كان لدى النساء المصابات بمتلازمة تكيس المبايض انخفاض كبير في مستويات القدرة الكلية لمضادات الأكسدة، والأديبونيكتين ( $P < 0.05$ )، مع زيادة كبيرة مصاحبة في مستويات الجلوكوز والأنسولين ومقاومة الأنسولين ( $P < 0.0001$ ) ومن المثير للاهتمام أنه لم يلاحظ أي ارتباطات ذات دلالة إحصائية بين المعلمات. **الاستنتاج:** وفقًا لذلك، خلصنا إلى أن انخفاض مستويات الأديبونيكتين والقدرة الإجمالية لمضادات الأكسدة جنبًا إلى جنب مع ارتفاع مستويات الجلوكوز والأنسولين ومقاومة الأنسولين قد توفر عوامل خطر تنبؤية لمتلازمة تكيس المبايض لدى النساء اللاتي يعانين من السمنة المفرطة مع أعراض أقل لمتلازمة تكيس المبايض.

**الكلمات المفتاحية:** متلازمة المبيض المتعدد الكيسات، القدرة الكلية لمضادات الأكسدة، مقاومة الأنسولين، الأديبونيكتين.