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## STUDY OF THE INVOLVEMENT OF ADIPONECTIN AND HOMA-IR IN TUNISIAN SUBJECTS WITH METABOLIC SYNDROME

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### Keywords:

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**ABSTRACT:** Metabolic syndrome (MS) is a multi-factorial disorder with insulin resistance as a major characteristic. Adiponectin, an adipocyte derived cytokine, can regulate glucose levels and lipid homeostasis by its insulin sensitizer properties. Low circulating levels of serum adiponectin has been reported as a risk factor for the development of MS. The aim of this case control study was to investigate the association of serum adiponectin concentration, insulin resistance and various risk factors with MS among Tunisian participants. We designed a case-control study involving 100 patients with MS (84 females and 16 males, mean age 54±11 years) and 100 healthy controls (60 females and 40 males, mean age 51±10 years). Serum total cholesterol and triglyceride were determined by enzymatic methods. Insulin resistance was calculated using the homeostasis model assessment (HOMA-IR) = fasting insulin (uU/ml) x fasting plasma glucose (mmol/l)/22.5 and serum adiponectin levels were measured by enzyme-linked immunosorbent assay. Interestingly, 72% of the subjects with MS had high blood pressure. Compared with controls, patients had significantly higher levels of triglycerides (1.64±0.88 mmol/l; 1.07±0.43 mmol/l; p=0.03) and lower levels of high-density lipoprotein cholesterol (1.26±0.41 mmol/l; 1.58±0.30 mmol/l; p=0.04). In addition, there was a significant difference in serum adiponectin levels between the subjects with MS and controls (13.96±5.23 µg/ml; 22.24±9.07 µg/ml; p=0.006) and the mean levels of HOMA-IR were 5.23±3.52 and 1.83±0.93 for patients and controls, respectively. In conclusion, hypoadiponectinemia and insulin resistance represent risk factors for MS development.

**INTRODUCTION:** Metabolic Syndrome (MS) is a cluster of lipid and metabolic abnormalities including central adiposity, hypertension, dyslipidemia and impaired glucose metabolism. It has been associated with increased risk of type 2 diabetes and cardiovascular disease (CVD) <sup>1, 2</sup>. MS is one of the most problem of world proportions affecting developed and underdeveloped countries in a rapidly progressive way <sup>3, 4</sup>.

In Tunisia, few studies have focused on the prevalence of MS <sup>5-7</sup>. On the other side, the prevalence of obesity is high and is increasing <sup>8</sup>. Obesity plays the most important role in the pathophysiology of the MS, a condition which is accompanied by insulin resistance (IR), hypertension and hyperlipidemia <sup>9</sup>. Interestingly, IR is defined as a state that requires more insulin to obtain the biological effects as achieved by a lower amount of insulin in the normal state.

In epidemiological studies, IR typically is quantified by the Homeostasis Model Assessment (HOMA) index. Indeed, we could previously show that HOMA-IR is associated with the MS but not with coronary artery disease (CAD) <sup>10</sup>. Adiponectin is a 244 amino acid protein secreted specifically by

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adipose tissue that has been recognized as a key regulator of lipid metabolism, endothelial function and insulin sensitivity<sup>11</sup>. Furthermore, low circulating levels of serum adiponectin was related to the development of MS and CVD<sup>9,12</sup>.

Dyslipidemia is well known as a major risk factor of MS and the role of high-density lipoprotein cholesterol (HDL-C) and triglycerides (TG) are already established as the key metabolic abnormalities in patients with IR<sup>13</sup>.

To our knowledge, in Tunisia, few studies have been investigated the relationship between lipid profile, insulin resistance and adiponectin with the risk of MS. Therefore, the purpose of this study was to explore the association of circulating adiponectin concentrations, insulin resistance and various risk factors with MS among Tunisian participants.

## **MATERIALS AND METHODS**

### **Study population:**

This study included 200 participants (100 subjects with MS and 100 others without MS) who are volunteers from external consultations from dispensary Kalaa Kebiraa, Sousse Tunisia. These subjects had no past history of CVD, no history of prescribed medicine. The main exclusion criteria were: pediatric study populations, age > 90 years old, several renal failures, an acute infection, thyroid problem or heart disease. This study was approved by the local Ethics Committee of Farhat Hachet Hospital of Sousse, Tunisia, in accordance with the Declaration of Helsinki.

### **Definition of Metabolic Syndrome:**

MS was defined as the presence of any three of the five following components according to the National Cholesterol Education Program Adult Treatment Panel III (NCEP ATPIII) criteria: abdominal obesity (defined as waist circumference  $\geq 102$  cm for men and  $\geq 88$  cm for women); TG  $\geq 150$  mg/dL (1.7 mmol/L); HDL-C  $< 40$  mg/dL (1.03 mmol/L) in men or  $< 50$  mg/dL (1.29 mmol/L) in women; fasting glucose  $\geq 100$  mg/dL (5.6 mmol/L) or medication for diabetes; and blood pressure  $\geq 130/85$  mmHg or on antihypertensive medication<sup>14</sup>.

### **Anthropometric measures and blood pressure:**

After obtaining a consent agreement from participant, weight and height were measured. Waist circumference (WC) was measured midway between the lowest rib and the iliac crest on standing subjects. Body Mass Index (BMI) was calculated as weight (kg) divided by height<sup>2</sup> (m). Arterial blood pressure was measured in participant rested and seated for at least 5 minutes using an automated blood pressure monitor.

### **Laboratory measurements:**

Blood was collected following overnight fasting, and plasma and serum samples were either used immediately for analysis or were stored frozen at  $-20^{\circ}\text{C}$ . Fasting blood glucose levels, urea, creatinine and non esterified fatty acids (NEFA) were measured with colorimetric essay (Randox-Antrim,UK; CX5 and CX9-BECKMANN).

The lipid profile included total cholesterol (TC), serum TG, HDL-C, apolipoprotein A1(Apo A1), apolipoprotein B(Apo B), lipoprotein Lp(a) as well as liver function including alanine aminotransferase (ALAT), aspartate transaminase (ASAT), gammaglutamyl transpeptidase (Gamma-GT), glycated hemoglobin (HbA1C), high-sensitivity C-reactive protein (hs\_CRP), uric acid, Cystatin C were performed using an autoanalyzer (COBAS Integra 400, Roche Diagnostics, Basel, Switzerland). Low density lipoprotein cholesterol (LDL-C) was determined by Friedewald equation<sup>15</sup>. Serum insulin and homocysteine concentrations were measured by an immunoassay (Abbott AxSYM-Diagnostics, Wiesbaden, Germany) and HOMA-IR were calculated using the formula =  $[\text{Fasting insulin } (\mu\text{U/ml}) \times \text{Fasting glucose (mmol/L)}] / 22.5$ <sup>16</sup>. Serum adiponectin levels were determined using a commercially available enzyme-linked immunosorbent assay (ELISA) kit (Abcsy Human Adiponectin ELISA).

### **Statistical evaluation:**

Statistical analyses were performed using SPSS software package version 18 .0 (SPSS Inc, Chicago, IL). Data are presented as mean  $\pm$  Standard deviation (SD) or the median and percentages. Means were compared using Student test. The significance threshold was set at 5% ( $P < 0.05$ ).

**RESULTS:****The clinical characteristics:**

**Table 1** lists the demographic and clinical characteristics of the population according to participants with and without the MS. Mean age was  $54 \pm 11$  years in the study group and  $51 \pm 10$

years for the healthy group. Among the cases, men/women ratio was 0.19, while in controls it was 0.66. Most of the subjects with MS had high blood pressure (72%), as this was one of the inclusion criteria. They had also higher BMI and WC than subjects without MS.

**TABLE 1: DEMOGRAPHIC AND CLINICAL CHARACTERISTICS OF THE SUBJECTS AMONG MS GROUP AND CONTROLS**

| Parameter                 | MS group (n=100) | Control group (n=100) |
|---------------------------|------------------|-----------------------|
| Age, years *              | 54±11            | 51±10                 |
| Men/women                 | 16 :84           | 40 :60                |
| BMI, Kg/m <sup>2</sup> *  | 32.21±5.23       | 25.52±2.94            |
| Waist circumference, cm * | 101.72±7.41      | 91.56±4.91            |
| Hypertension, %           | 72               | 0                     |
| Sedentary, %              | 42               | 20                    |
| Smoking, %                | 11               | 19                    |
| Alcohol consumption, %    | 5                | 0                     |

\*Values expressed as mean standard deviation BMI denotes body mass index

**The laboratory variables:**

**Table 2** shows the general laboratory data of the 200 subjects included in this study. In contrast to participants without MS, those with MS had significantly higher fasting blood glucose, TG, creatinine, uric acid and cyctatin C. The MS group

showed also significantly higher mean hs-CRP, NEFA and homocystein and lower HDL-C levels than the control group. However, there was no significant difference in CT, LDL-C, ApoA1, ApoB and Lp (a) between the two groups.

**TABLE 2: BIOCHEMICAL DATA OF THE SUBJECTS AMONG MS GROUP AND CONTROLS**

| Variables                      | MS group (n=100)    | Control group (n=100) | P value |
|--------------------------------|---------------------|-----------------------|---------|
| Fasting plasma glucose, mmol/l | 8.46±3.74           | 5.10±2.12             | 0.000   |
| Fasting serum insulin, µU/ml   | 13.62±5.76          | 8.20±4.13             | 0.000   |
| HbA1C, %                       | 7.56±2.87           | 5.87±0.81             | 0.005   |
| Urea, mmol/l                   | 5.72±2.64           | 4.37±1.22             | 0.229   |
| Creatinine, µmol/l             | 75.13±59.78         | 64.73±19.73           | 0.038   |
| Uric acid, µmol/l              | 231±106.49          | 208.30±63.35          | 0.021   |
| Cyctatin-c, mg/l               | 0.77±0.21           | 0.55±0.23             | 0.05    |
| TC, mmol/l                     | 5.68±1.84           | 5.14±1.16             | 0.064   |
| TG, mmol/l                     | 1.64±0.88           | 1.07±0.43             | 0.03    |
| HDL-C, mmol/l                  | 1.26±0.41           | 1.58±0.30             | 0.04    |
| LDL C, mmol/l                  | 3.68±1.48           | 3.27±1.10             | 0.318   |
| Apo A1, g/l                    | 1.33±0.17           | 1.62±0.36             | 0.193   |
| Apo B, g/l                     | 0.89±0.27           | 0.84±0.17             | 0.234   |
| Lp(a), g/l                     | 0.52±0.33           | 0.40±0.34             | 0.738   |
| Gamma-GT, UI/L                 | 20.07±10.28         | 15.89±4.76            | 0.237   |
| ASAT, UI/L                     | 22.32±6.68          | 20.17±7.44            | 0.577   |
| ALAT, UI/L                     | 23.63±13.24         | 18.24±5.14            | 0.049   |
| Hs-CRP                         | 2.690 (1.525-5.320) | 2.10 (1.760-2.800)    | 0.01    |
| NEFA, mmol/l                   | 0.56±0.22           | 0.40±0.09             | 0.04    |
| Homocysteine, µmol/l           | 12.35±4.28          | 9.04±2.08             | 0.034   |

Abbreviations: alanine aminotransferase (ALAT), aspartate transaminase(ASAT), apolipoprotein A1(Apo A1), apolipoprotein B(Apo B), gammaglutamyl transpeptidase (Gamma-GT), glycated hemoglobin (HbA1C), high density lipoprotein cholesterol (HDL-C), high-sensitivity C-reactive protein (hs\_CRP), lipoprotein Lp(a), low density lipoprotein cholesterol (LDL-C), metabolic syndrome (MS), non esterified fatty acids (NEFA), total cholesterol (TC), triglycerides(TG), Data presented are mean±standard deviation. Data for hs-CRP were presented as median (interquartile range).

**Evaluation of circulating adiponectin levels and insulin-resistance index:**

HOMA-IR scores were found significantly higher ( $p=10^{-3}$ ) in MS group ( $5.23\pm 3.52$ ) than in the control group ( $1.83\pm 0.93$ ). The values of serum adiponectin were found abnormal in MS group ( $13.96\pm 5.23$ ) compared to the control ( $22.24\pm 9.07$ ) and statistical differences were found between the two groups ( $p=0.006$ ).

**DISCUSSION:**

In the present study, the prevalence of MS was higher in women than in men because of their higher frequency of abdominal obesity. These results confirmed previous findings in Tunisian population<sup>5-7, 17</sup> as well as in others population<sup>18-21</sup>. Sedentary lifestyle, the lack of participation in physical activities among adult Tunisian women, hormonal factors and postmenopausal weight gain may prove the large waist circumference in women<sup>22, 23</sup>. We found also that serum adiponectin levels were negatively associated with MS and we showed that insulin resistance as assessed by the HOMA index is significantly linked with the MS.

Adipocyte is an active endocrine secretory cell that release several cytokines including tumor necrosis factors alpha, visfatin, leptin, interleukins and adiponectin<sup>24</sup>. In contrast to the inflammatory adipokines, adiponectin increases insulin sensitivity and regulates angiogenic and endothelial function<sup>25</sup>. Several studies have demonstrated that serum adiponectin level is inversely associated with obesity<sup>26, 27</sup> and insulin resistance<sup>28-30</sup>. Hypoadiponectemia is significantly related to type 2 diabetes<sup>31</sup>, and independently associated with the MS<sup>12, 32, 33</sup>. These findings are in agreement with those of the present study which revealed that circulating adiponectin levels were inversely associated with the presence of MS.

Liang et al. demonstrated that circulating adiponectin level was lower in subjects with combined MS and cardiac syndrome X (CSX) than those without MS and CSX. It has also been reported in this study that IR index was significantly higher in subjects with combined MS and CSX than the control group<sup>9</sup>. Pathophysiologically, IR is the key feature of MS<sup>10</sup> and many studies mentioned that there is a close

relationship between MS and IR<sup>30, 34</sup>. Our study confirms these findings by showing that HOMA-IR showed significant positive correlation with MS.

In other hand, M. Eslamian et al. showed that lipid profile was strongly correlated with blood concentration of adiponectin in patients with type 2 diabetes and MS<sup>35</sup>. It was proved that adiponectin influences plasma lipoprotein levels by altering the levels and activity of key enzymes (lipoprotein lipase and hepatic lipase) responsible for the catabolism of triglyceride-rich lipoproteins and HDL-C<sup>36</sup>. Several studies found that serum adiponectin levels were correlated positively with HDL-C concentration and negatively with TG concentration<sup>12, 35</sup>, and the present results showed that low level of adiponectin was associated with higher TG levels and lower HDL-C in Tunisian subjects with MS.

Additionally, our cases showed higher uric acid levels which is in accordance with Nejatinamini et al.<sup>37</sup> who reported that uric acid and MS components supports might be an additional components of MS. Uric acid may not only acts as surrogate marker of MS, but also have a pathogenic role in the development of MS, and therapies that lower uric acid may improve certain components of MS<sup>38</sup>. Although hyperuricemia is recognized as a risk factor for obesity, diabetes and MS, the precise physiological mechanisms by which uric acid contribute to these diseases state remain unclear<sup>39</sup>. Inflammation plays an important role in the pathogenesis of MS, hs-CRP has been found to be associated with MS<sup>40</sup>. Our study showed that the concentrations of hs-CRP were higher in subjects with MS than those without MS.

The limitations of this study include its relatively small sample size, thus there might be effects on generalization of our findings and the cross-sectional design limited our ability to infer a causal relation between adiponectin concentration and development of MS.

In conclusion, our results illustrate a negative association between circulating fasting adiponectin and MS among Tunisian participants. Lipid profile, uric acid, Hs-CRP and insulin resistance are significantly associated with MS.



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