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PREVALENCE AND RISK FACTORS OF METABOLIC SYNDROME IN ADULT TUNISIAN PATIENTS

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ABSTRACT: Background: Metabolic syndrome (MetS) is a cluster of diseases, including hypertension, obesity, dyslipidemia, and hyperglycemia. We therefore aimed to estimate MetS prevalence and to determine risk factors for MetS. **Methods:** In the present study, we recruited 209 adult Tunisian MetS patients (57 males and 152 females, mean age 58.9±10.7 years) matched by gender and ethnic origin to 193 healthy individuals (31 males and 162 females, mean age 50.3±10.6 years). All subjects underwent anthropometric and biochemical examinations. **Results:** Interestingly, the prevalence of MetS was 21.89% in males and 78.1% in females. Notable increase in the dyslipidemia (64.1%), hypertension (77%), and obesity (83.73%) in patients with MetS were seen. In addition, insulin resistance, hyperglycemia, abnormal lipid profiles, increased free fatty acids, and lower serum adiponectin concentrations were all associated with MetS. **Conclusion:** We suggest that the MetS is highly prevalent among patients group than healthy subjects. Modification of lifestyle factors, even later in life, has considerable potential for primary prevention of the MetS.

INTRODUCTION: Metabolic syndrome (MetS) is a collection of cardiometabolic risk factors that includes obesity, insulin resistance, hypertension, and dyslipidemia^{1, 2}. It has been proposed that this syndrome is closely associated with lifestyle³ and is a powerful determinant of diabetes and cardiovascular diseases (CVD). It has been established that those with MetS have a five times higher risk of developing T2DM and three times higher risk of developing CVD⁴. Due to the collection of different components, several organizations have established different diagnostic criteria for MetS.

In addition, a great deal of attention has been focused on MetS in the adult population. On the basis of the most recent epidemiological analysis using the American Heart Association/National Heart, Lung, and Blood Institute 2005 guidelines⁵, similar to those of National Cholesterol Education Program/Adult Treatment Panel III⁶, approximately 1 adult in every 4 or 5 could be characterized as having the MetS.

This incidence increases with age; it has been estimated that in people over 50 years of age. A higher percentage (40.1%) of prevalence occurred with revised International Diabetes Federation 2005 criteria, which use a lower cutoff point for waist (≥ 94 cm in men and ≥ 80 cm in women). These elevated rates indicate the existence of a problem that merits investigation, since it is known that in the majority of cases MetS can be avoided by identification of risk factors and initiation of preventative measures.

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Furthermore, MetS is a complex phenotype that correlates with many clinical conditions besides CVD and diabetes, including chronic low-grade inflammation, oxidative stress⁷, hyperuricemia⁸, hypertension⁹, dyslipidemia⁹, hyperandrogenism¹⁰, hepatic steatosis¹¹, nonalcoholic fatty liver disease (NAFLD)¹², impaired glucose tolerance¹³, hypogonadism¹⁴, and vascular dementia¹⁵.

Therefore, we conducted a case control to examine whether the individual components of MetS and to identify the correlation of the different risk factors for development of the MetS with cardio-metabolic parameters and insulin resistance, and its identification may thus be important in the risk assessment and treatment of the patients.

MATERIAL AND METHODS:

Subjects: This is a prospective study, in which sampling was carried between January and September 2014. All participants gave written informed consent and the Hospital's Ethics Committee approved the study protocol.

The study is based on two types of populations recruited from intermediate group of basic health Sousse Tunisia: a control population and population with MetS. The healthy subjects, recruited to attend for a routine check-up (in the same time of patients recruitment), included 31 male and 162 females, with a mean age of 58±12.5 years and 48.82±9.66 years, respectively. Those with medical illnesses such as acute infection, chronic renal failure, malignancies, and other severe medical illnesses were excluded from the enrollment. Alcohol intake, smoking habits, medication history, and medical history, at the time of coronary angiograms, were obtained by the chart review and from the self-questionnaire.

The patients included 57 males and 152 females, with a mean age of 60.11±10.71 years and 58.48±10.83 years, respectively.

MetS and metabolic risks are defined according to the US National Cholesterol Education Program Adult Treatment Panel III (NCEP ATPIII) guidelines. MetS was defined as having at least three of the following metabolic risk factors:

- Central obesity (waist circumference (WC) > 88 cm in women, > 102 cm in men);

- Hypertriglyceridemia (fasting triglycerides \geq 1,7 mmol/l (1,50 g/l));
- Low high-density lipoprotein (HDL) cholesterol (fasting HDL <1.29 mmol/l (0,50 g/l) in women, < 1,03 mmol/l (0,40 g/l));
- Glucose intolerance (fasting glucose \geq 6,1 mmol/l (1,10 g/l));
- Hypertension (sitting blood pressure \geq 130/85 mmHg obtained as a mean of two readings taken after resting for at least 10 min or on regular antihypertensive medications).

Measurements: Height, weight, systolic, and diastolic blood pressures were measured in duplicate and the results were averaged. Weight height and waist circumference were measured. The body mass index (BMI) was calculated by dividing the weight (kg) with the square of height (m).

All blood samples were obtained after overnight fasting. Fasting blood glucose, urea and creatinine were measured from fluoride plasma. Total cholesterol, triglyceride, high-density lipoprotein cholesterol (HDL-C), uric acid, aspartate aminotransferase (AST), alanine aminotransferase (ALT), α -glutamyltransferase (α GT) and free fatty acids (FFA) were measured. Low-density lipoprotein cholesterol (LDL-C) was calculated using the Friedewald equation: [LDL-C (mmol/l) = CT - (TG / 2.2 + HDL-C)]. Serum insulin was determined with a microparticle enzyme immunoassay. The insulin resistance index estimated by the homeostasis model assessment method (Homeostasis Model Assessment – Insulin Resistance (HOMA-IR)) was calculated as follows: [fasting serum insulin (μ U/ml) x fasting plasma glucose (mmol/l)/22.5].

Those patients were well controlled in terms of glycosylated hemoglobin (HbA1c) determined by a latex agglutination immunoassay type TINIA (turbidimetric inhibition immunoassay) by automate (Randox, Antrim, UK). High-sensitivity C-reactive protein (Hs-CRP), apolipoprotein A (Apo A), apolipoprotein B (Apo B), lipoprotein (a) (Lp (a)) and cystatin C were measured from the samples by immunoturbometric COBAS. Homocysteine was measured from samples by AXSYM ABBOTT.

Serum adiponectin levels were determined using a commercially available enzyme-linked immunosorbent assay (ELISA) kit (Biosource-Invitrogen Corporation, Carlsbad, CA).

Statistical Analysis: Database management and statistical analyses were carried out using SPSS (Statistical Package for the Sociological Sciences), version 18.0. Results are presented as means ± SD, or percentages. Means were compared using Student test. The relations between variables were assessed with Pearson’s correlation analysis. The significance threshold was set at 5% (p<0.05).

RESULTS: The study shows that the prevalence of MetS according to NCEP-ATP III definition was 21.89% in males and 78.1% in females. Of all

patients, 73.20% were obese, and obesity was more frequent among females (P < 0.05). Mean body mass index was 30.85±5.01. Out of 88 males and 314 females, hypertension was observed as 22.48% and 54.52%, respectively, in the metabolic syndrome population, which was found to be statistically significant (p < 0.05). The percentage of high triglyceride and low HDL-C levels in the total MetS population was 40.66% and 70.33% respectively; in males this was 11.96% and 15.79%, and 28.70% and 54.54% in females, respectively. Fasting glucose was observed as 18.18% and 55.02% in males and females, respectively (P < 0.05). The frequencies of MetS risk factors diagnosed by NCEP-ATPIII are given in **Table 1**.

TABLE 1: FREQUENCY OF GLUCOSE INTOLERANCE, HIGH WAIST CIRCUMFERENCE, HIGH BLOOD PRESSURE, ELEVATED TG, AND LOW HDL-C IN MetS PATIENTS DIAGNOSED BY NCEP ATPIII CRITERIA.

	Glucose intolerance	Obesity	Hypertension	HyperTG	Low HDL-C
MALE	38(18.18%)	27(12.92%)	47(22.48%)	25(11.96%)	33(15.79%)
FEMALE	115(55.02%)	148(70.81%)	114(54.52%)	60(28.70%)	114(54.54%)
TOTAL	153(73.20%)	175 (83.73%)	161 (77%)	85(40.66%)	147(70.33%)

TG: triglycerides; HDL-C: high-density

Table 2 shows the clinical and metabolic characteristics of subjects with and without MetS according to ATPIII diagnostic criteria. Of the 402 subjects, 209 were diagnosed as having metabolic syndrome. These patients had significantly higher levels of waist circumference, BMI and

hypertension which were found to be statistically significant (p < 0.05). **Table 2** puts in consideration the percentage of Alcohol intake, smoking habits, medication history, medical history, diet and sedentarity, of the study population, for their role that affect the management of MS.

TABLE 2: CLINICAL CHARACTERISTIC OF SUBJECTS WITH METABOLIC SYNDROME COMPARED TO SUBJECTS WITHOUT METABOLIC SYNDROME

Risk factors	MetS group (n= 209)	Control group (n= 193)	P
Number of subjects	209	193	-
Age (years)	58.9±10.7	50.3±10.6	0.026
Male /female	57/152	31/162	0.006
Waist circumference (cm) Male	102.54±11.34	93.23±10.60	0.067
Female	102.06±9,12	93.90±12.01	0.046
BMI (Kg/m ²)	30.85±5.01	26.93±4.68	0.014
Hypertension (%)	77	0	0.003
Smoking (%)	8.1	0	<10 ⁻³
Alcohol (%)	5	0	NS
Treatment (%)	47.8	0	0.002
Diet (%)	37.8	0	0.047
Physical inactivity (%)	31.1	0	0.058
Antecedents (%)	52.2	28.1	<10 ⁻³

Table 3 shows the clinical profile between the patients with and without MetS. Of the 209 patients, 57 male and 152 female were diagnosed

as having metabolic syndrome. These patients had significantly higher levels of fasting glucose, HbA1c, HOMA-IR, fasting insulin, triglycerides,

total cholesterol, LDL-C, Apo B, Lp (a), urea, uric acid, cystatin C, GGT, ASAT, ALAT, homocysteine, CRPus, and free fatty acids, but significantly lower concentrations of adiponectin,

HDL-C and Apo A1, as compared with those without MetS. No significant difference was observed in creatinine between the 2 groups.

TABLE 3: BIOLOGICAL CHARACTERISTICS OF SUBJECTS WITH METABOLIC SYNDROME INCLUDED IN THE STUDY

Parameters	MetS group (n= 209)	Control group (n= 193)	P
Fasting glucose (mmol/l)	8.52 ± 3.94	5.18 ± 0.65	<10 ⁻³
HbA1C (%)	7.94 ± 2.25	6.29 ± 0.97	0.047
Fasting insulin (uU/ml)	13.61±5.74	8.26±2.67	0.002
HOMA-IR	5.22±3.72	1.90±0.67	<10 ⁻³
Total cholesterol (mmol/l)	5.43±1.61	4.70±1.07	<10 ⁻³
HDL-C (mmol/l)	1.16±0.36	1.36±0.35	<10 ⁻³
LDL-C (mmol/l)	3.50±1.35	2.87±0.89	<10 ⁻³
Triglycerides (mmol/l)	1.68±0.92	1.008±0.37	10 ⁻³
Apo A1 (g/l)	1.30±0.37	1.69±0.29	<10 ⁻³
Apo B (g/l)	1.09±0.26	0.90±0.23	<10 ⁻³
Lp (a) (g/l)	0.39±0.34	0.21±0.14	<10 ⁻³
urea (mmol/l)	5.83±2.52	4.94±1.83	<10 ⁻³
creatinine (µmol/l)	74.63±42.12	69.60 ±23.86	NS
uric acid (µmol/l)	235±95.23	201.63±69.71	<10 ⁻³
Cystatin C (mg/l)	1.13±0.38	0.77±0.16	0.006
GGT (IU/L)	18.97±9.20	16.50±5.70	0.01
ASAT (IU/L)	20.99±7.83	19.56 ±7.009	0,054
ALAT (IU/L)	20.15±9.44	17.92±7.83	0.01
Adiponectin (mg/l)	12.21±7.58	19.93±7.95	0.042
Homocysteine (µmol/l)	13.70±5.76	9.92±3.49	<10 ⁻³
CRPus (mg/l)	5.34±5,61	1.97±1,17	<10 ⁻³
Free fatty acids (µmol/l)	0.92±0.24	0.34±0.15	<10 ⁻³

Abbreviations: glycosylated hemoglobin (HbA1c), Homeostasis Model Assessment – Insulin Resistance (HOMA-IR), High density lipoprotein cholesterol (HDL-C), Low density lipoprotein cholesterol (LDL-C), apolipoprotein A1 (apoA1), apolipoprotein B (apoB), lipoprotein (a) (Lp (a)), aspartate aminotransferase (AST), alanine aminotransferase (ALT), γ -glutamyltransferase (γ GT) highsensitivity C-reactive protein (hs-CRP).

Levels of FFA, after adjustment for age, gender, and BMI, were positively correlated with TG, FBS, hs-CRP and HOMA-IR, but inversely correlated with HDL-C and its main component apoA1.

Serum adiponectin concentrations, after adjustment for above parameters, were positively correlated with HDL-C and ApoA1 but inversely correlated with TG, FBS, hs-CRP and HOMA-IR (**Table4**).

TABLE 4: CORRELATION OF SERUM ADIPONECTIN AND FREE FATTY ACIDS CONCENTRATIONS WITH CARDIO-METABOLIC PARAMETERS AND INSULIN RESISTANCE MEASURED USING THE HOMEOSTASIS MODEL ASSESSMENT OF INSULIN RESISTANCE (HOMA-IR).

	TG	HDL-C	ApoA1	FBS	hs-CRP	HOMA-IR
FFA	0.178	-0.168	-0.126	0.115	0.135	0.166
Adiponectin	-0.167	0.127	0.092	-0.119	-0.109	-0.154

TG: triglycerides; HDL-C: high-density lipoprotein cholesterol; apoA1: apolipoprotein A1; FBS: fasting glucose; hs-CRP: highsensitivity C-reactive protein; HOMA-IR: Homeostasis Model Assessment – Insulin Resistance; FFA:

DISCUSSION: From this study we have found that the prevalence of MetS in the studied population was 52% based on NCEP-ATPIII criteria. Kaur et al¹⁶ reported that the worldwide prevalence of MetS to be between 10 and 84% depending on the ethnicity, age, gender and race of

the population, whereas the IDF estimates that one-quarter of the world's population has MetS. According to Pal and Ellis¹⁷ 20% of adults in the Western world have MetS.

Some studies show that MetS is more prevalent among females than males¹⁸⁻²² and another studies show that the prevalence of MetS is less prevalent among males than females²³, but other studies show that the prevalence of MetS was similar among males and females²⁴⁻²⁶. In this study the MetS is more prevalent among females than males (21.89% in males and 78.1% in females).

In the United States, the prevalence of MetS was 21.8% using the ATP III definition²⁵. The prevalence was similar for male (24.0%) and female (23.4%) subjects. The prevalence in Isfahan (Iran) was 65.0% with higher rate in females than males (71.7% female and 55.8% male)²⁷. The prevalence in Karachi (Pakistan) was 79.7% in type 2 diabetics (45.5% females and 34.3% males)²⁸. The overall prevalence of MetS in Japanese type 2 diabetic patients was 168 (26.37%) out of 637 type 2 diabetic patients. The prevalence was higher in males (45.9%) than females (28.0%)²⁹. Another study done in Korean estimates the overall prevalence was 32.6%. The prevalence was found to be 46.9% and 65.1% among males and females respectively³⁰. The overall prevalence among Saudis with type 2 diabetes was 22.64% (19.49% male, 25.17% female)³¹.

The study done in the Gorgan, Golestan province (South East of Caspian Sea), Iran was the higher prevalence of MetS among females (84.70% and 88.23%) when compared with males (62.60% and 60.97%), and that the prevalence of MetS in Gorgan is appreciably higher compared with that in some other countries. Females were more affected than males³². This may be due to the specific characteristics in the lifestyle changes between females and males. The majority of females with MetS were householder. It seems that they do less physical activity at home³³.

Additionally, we reported hypertension were significantly prevalent ($p < 0.005$) in 22.48% of males and in 54.52% of females. In this study, 58% of the participants had high Waist circumference. Our results show, that on moving from baseline to higher levels in hypertension, triglyceride, and HDL-C, the crude relative prevalence in each parameter has increased significantly. The present study corroborates with the observations of previous studies³⁰⁻³³. Over half of the participants

had hypertension and dyslipidemia, which is in accordance with the previously reported studies²⁶. We found an overall prevalence of hyperglycemia in the studied population as 73.20%.

Moreover, obesity is an important, easily observed, and measurable risk factor for the MetS³⁴. BMI and weight gain remained strong significant predictors of this syndrome. It is not surprising given evidence from longitudinal observational studies that higher BMI and weight gain over time are associated with poorer blood pressure, higher fasting glucose, and dyslipidemia—the remaining components of the syndrome³⁴. A multiethnic representative US sample of 12,363 men and women 20 years and older from the third National Health and Nutrition Examination Survey (NHANES) were evaluated for MetS as defined by the ATP III diagnostic criteria and the disorder was found to be present in 22.8% and 22.6% of the men and women, respectively. MetS was present in 4.6%, 22.4%, and 59.6% of normal-weight, overweight, and obese men, respectively, and physical inactivity was associated with an increased risk of developing the syndrome³⁵. In obese individuals, the prevalence of MetS is about 40%³⁶. In our study, of all patients 73.20% were obese, and obesity was more frequent among females (12.92% male and 70.81% female). Insulin resistance and obesity play a central role in causing hypertension and dyslipidemia, and further predisposes to MetS^{37,38}.

Individual components of MetS such as HDL-C, systolic hypertension, and other known traditional risk factors (sex, total cholesterol) were significant predictors of future coronary heart disease (CHD) events. Klein et al.³⁹ reported that high-risk lipid levels (either high serum total cholesterol or low HDL-C, or high ratio of these two levels) occurred most commonly in the combinations of MetS components that were associated with increased CVD risk⁴⁰. Elevated triglyceride levels may be secondary to hyperglycemia⁴¹.

In our study, FFA and adiponectin were correlated with cardio-metabolic parameters. We found a positive correlation between FFAs and insulin resistance, as determined by HOMAIR. Insulin resistance and FFAs may have a cause and effect relationship⁴².

Interestingly, in our study, adiponectin was inversely correlated with FBS, hs-CRP and HOMA-IR. In humans, decreased plasma adiponectin levels have been demonstrated in patients with obesity, diabetes, and CHD⁴³⁻⁴⁵. Moreover, the degree of hypoadiponectinemia has been reported to be correlated with the degree of insulin resistance^{46, 47}, and hypoadiponectinemia has been shown to be closely associated with the clinical phenotype of metabolic syndrome^{48, 49}. Adiponectin served as an antiinflammatory molecule for vascular walls as well as adipose tissue⁵⁰, hypoadiponectinemia was observed in obesity-linked diseases^{43, 51}. Therefore, hypoadiponectinemia can be responsible for a low-grade systemic chronic inflammation state, which is closely related to an increased hs-CRP level. Adiponectin level may be a convenient and sensitive biomarker for the prediction of insulin resistance and MetS^{52, 53}. The MetS is reported to be one of the conditions associated with insulin resistance and/or atherosclerosis in humans^{51, 54}.

CONCLUSION: In conclusion, the prediction of cardiovascular risk factors called the MetS, based on the NCEP ATPIII definition, defined by the presence of a cluster of metabolic abnormalities, including impaired glucose metabolism, high BMI and abdominal fat distribution, dyslipidemia, hypertension, and insulin resistance. The presence of the metabolic syndrome was associated with an increased risk for cardiovascular morbidity and mortality⁵⁵ and its identification may thus be important in the risk assessment and treatment of the patients with MetS in Tunisian population and will be pivotal in the prevention of CVD and type 2 diabetes.

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CONFLICTS OF INTEREST: The authors declare that they have no conflict of interest.

REFERENCES:

- Sookoian S, Pirola C. Metabolic syndrome: from the genetics to the pathophysiology. *Curr Hypertens Rep* 2011; 13: 149–157.
- Grassi G, Seravalle G, Quarti-Trevano F, Dell'Oro R, Bombelli M, Mancia G. Metabolic syndrome and cardiometabolic risk: an update. *Blood Press*. 2009; 18(1-2):7-16.
- Yamaoka K and Tango T: Effects of lifestyle modification on metabolic syndrome: a systematic review and meta-analysis. *BMC Medicine* 2012; 138:1741-7015.
- Sookoian S, Pirola C. Metabolic syndrome: from the genetics to the pathophysiology. *Curr Hypertens Rep* 2011; 13: 149–157.
- Grundy SM. Metabolic Syndrome Scientific Statement by the American Heart Association and the National Heart, Lung, and Blood Institute. *Arteriosclerosis, Thrombosis, and Vascular Biology*. 2005; 25: 2243-2244.
- Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults, executive summary of the third report of the National Cholesterol Education Program (NCEP). Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults Adult Treatment Panel III. *JAMA* 2001; 285:2486–97.
- Van Guilder GP, Hoetzer GL, Greiner JJ, Stauffer BL, DeSouza CA. Influence of Metabolic Syndrome on Biomarkers of Oxidative Stress and Inflammation in Obese Adults. *Obesity*. 2006; 12: 2127–2131.
- Changuia L, Ming-Chiab H, Shun-Jenc C. Metabolic syndrome, diabetes, and hyperuricemia. *Current Opinion in Rheumatology* 2013; 25: 210–216.
- Yadav D, et al., Prevalence of Dyslipidemia and Hypertension in Indian Type 2 Diabetic Patients with Metabolic Syndrome and its Clinical Significance, *Osong Public Health Res Perspect* 2014 ;5(3): 169–175.
- Özdemir S, Özdemir M, Görkemli H, Kiyici A, Bodur S: Specific dermatologic features of the polycystic ovary syndrome and its association with biochemical markers of the metabolic syndrome and hyperandrogenism. *Acta Obstetrica et Gynecologica Scandinavica* 2009; 89(2): 199-204.
- Dekker MJ, Su Q, Baker C, Rutledge AC, Khosrow A. Fructose: a highly lipogenic nutrient implicated in insulin resistance, hepatic steatosis, and the metabolic syndrome. *American Journal of Physiology - Endocrinology and Metabolism* 2010; 299 (5): 685-694.
- Paschos P, Paletas K. Non alcoholic fatty liver disease and metabolic syndrome. *Hippokratia* 2009; 13 (1): 9-19.
- Moran LJ, Misso ML, Wild RA, Robert J, Norman RJ: Impaired glucose tolerance, type 2 diabetes and metabolic syndrome in polycystic ovary syndrome: a systematic review and meta-analysis. *Human Reproduction Update* 2010; 16(4):347–363.
- Naifar M, Rekik N, Messedi M, Lahiani A, Turki M, Abid M, Avedi F, Jamoussi K: Male hypogonadism and metabolic syndrome. *Andrologia* 2015; 47(5):579–586.
- Raffaitin C, Gin H, Empana, JP and al. Metabolic Syndrome and Risk for Incident Alzheimer's disease or Vascular Dementia. *Diabetes Care* 2009; 32(1): 169-174.
- Kaur JA. Comprehensive review on metabolic syndrome. *Cardiol Res Pract* 2014; 2014: 943162.
- Pal S, Ellis V. The chronic effects of whey proteins on blood pressure, vascular function, and inflammatory markers in overweight individuals. *Obesity (Silver Spring)* 2010; 18: 1354–1359.
- Zhao Y, Yan H, Yang R et al. Prevalence and determinants of metabolic syndrome among adults in a rural area of Northwest China. *PLoS ONE* 2014; 9: e91578.
- Lydia UK, Yeri Kombe, MD and al. Prevalence of Metabolic Syndrome among an Urban Population in Kenya. *Diabetes Care* 2012; 35(4): 887-893.
- Gupta R, Deedwania PC, Gupta A, Rastegar S, Pan war RB, Kithara K. Prevalence of metabolic syndrome in an Indian urban population. *Int J Cardiol* 2004; 97:257–61.
- Mycolic R, Bianchi C, Bodyguard L, Pennon G, Caricature F, Giovannitti MG, et al. Prevalence of the metabolic syndrome among Italian adults according to ATPIII definition. *Nutr Metab Cardiovasc Dis* 2005; 15:250–4.
- Park HS, Oh SW, Cho SI, Choi WH, Kim YS. The metabolic syndrome and associated lifestyle factors among South Korean adults. *Int J Epidemiol* 2004; 33:328–36.

23. Sawant A, Mankeshwar R, Shah S et al. Prevalence of metabolic syndrome in urban India. *Cholesterol* 2011; 2011: 920983.
24. Moreira GC, Cipullo JP, Ciorlia LAS, Cesarino CB, Vilela-Martin JF. Prevalence of Metabolic Syndrome: Association with Risk Factors and Cardiovascular Complications in an Urban Population. *PLoS ONE* 2014; 9(9): e105056.
25. Ford ES, Giles WH, Dietz WH. Prevalence of the metabolic syndrome among US adults: findings from the Third National Health and Nutrition Examination Survey. *J Am Med Assoc* 2002; 287:356-9.
26. Yadav D, et al., Prevalence of Dyslipidemia and Hypertension in Indian Type 2 Diabetic Patients with Metabolic Syndrome and its Clinical Significance, *Osong Public Health Res Perspect* 2014; 5(3): 169-175.
27. Janghorbani M, Amini M. Metabolic syndrome in type 2 diabetes mellitus in Isfahan, Iran: prevalence and risk factors. *Metab Syndr Relat Disord* 2007; 5: 243-54.
28. Imam SK, Shahid SK, Hassan A, Alvi Z. Frequency of the metabolic syndrome in type 2 diabetic subjects attending the diabetes clinic of a tertiary care hospital. *J Pak Med Assoc* 2007; 57:239-42.
29. Yoshinori S, Keiko T, Machi F, Yoshiaki K, Shyoichi Y, Kishio N, et al. Prevalence of metabolic syndrome in Japanese type 2 diabetic patients and its significance for chronic vascular complications. *Diabetes Res Clin Pract* 2008; 79: 310-7.
30. Kim WY, Kim JE, Choi YJ, Huh KB. Nutritional risk and metabolic syndrome in Korean type 2 diabetes mellitus. *Asia Pac J Clin Nutr* 2008; 17:47-51.
31. Almoutaz Alkhier A. The prevalence of metabolic syndrome among type 2 Saudi diabetic patients a particular view in Gurayat Province 2008; 6(7):3-7.
32. Marjani A, Shirafkan A. The metabolic syndrome in type 2 diabetic patients in Gorgan: According to NCEP ATP III and IDF definitions. *Diabetes Metab Syndr*. 2011; 5(4):207-10.
33. Strasser B. Physical activity in obesity and metabolic syndrome. *Ann N Y Acad Sci*. 2013; 1281:141-59.
34. Ma X, Zhu S. Metabolic syndrome in the prevention of cardiovascular diseases and diabetes—still a matter of debate? *Eur. J. Clin. Nutr.* 2013; 67 (5):518-521.
35. Flegal KM, Carroll MD, Ogden CL, Curtin LR. Prevalence and trends in obesity among US adults, 1999-2008. *JAMA* 2010; 303: 235-241.
36. Termizy HM, Mafauzy M. Metabolic syndrome and its characteristics among obese patients attending an obesity clinic. *Singapore Med J* 2009; 50(4):390-4.
37. Allison M, Elizabeth PN, Marjka B, Linda CT. Identifying metabolic syndrome in a clinical cohort: Implications for prevention of chronic disease. *Preventive Medicine Reports* 2016; 4:502-506.
38. Gallagher EJ, Leroith D, Karnieli E. Insulin resistance in obesity as the underlying cause for the metabolic syndrome. *Mt Sinai J Med*. 2010; 77(5):511-23.
39. Klein BEK, Klein R, Lee KE. Components of the metabolic syndrome and risk of cardiovascular disease and diabetes in Beaver Dam. *Diabetes Care* 2002; 25:1790-4.
40. Protosaltis I et al. Metabolic syndrome and its components as predictors of all-cause mortality and coronary heart disease in type 2 diabetic patients *Atherosclerosis* 2007; 195 189-194.
41. Taskinen MR, Lahdenperä S, Syvännä M: New insights into lipid metabolism in non-insulin-dependent diabetes mellitus. *Ann Med* 1996; 28:335-340.
42. Ormseth MJ et al. Free Fatty Acids are Associated with Metabolic Syndrome and Insulin Resistance, but not Inflammation in SLE Patients. *Lupus*. 2013; 22(1): 26-33.
43. Ersilia N, Olga S, Maria LM. New Insight into Adiponectin Role in Obesity and Obesity-Related Diseases. *BioMed Research International* 2014; 14: 658913.
44. Hotta K, Funahashi T, and al: Plasma concentrations of a novel, adipose-specific protein, adiponectin, in type 2 diabetic patients. *Arterioscler Thromb Vasc Biol* 2000; 20:1595-1599.
45. Liu M and Liu F. Up- and down-regulation of adiponectin expression and multimerization: mechanisms and therapeutic implication. *Biochimie* 2012; 94: 2126-2130.
46. Rosa A, Ludovica M, Capasso M et al. Adiponectin oligomers as potential indicators of adipose tissue improvement in obese subjects. *European Journal of Endocrinology* 2013; 169:37-43.
47. Kawamoto R, Tabara Y, Kohara K, Miki T, Kusunoki T, Takayama S and al. Relationships between lipid profiles and metabolic syndrome, insulin resistance and serum high molecular adiponectine in Japanese community-dwelling adults. *Lipids in Health and Disease* 2011; 10:79.
48. Calton EK, Miller VS, and Soares MJ. Factors determining the risk of the metabolic syndrome: is there a central role for adiponectin. *The European Journal of Clinical Nutrition* 2013; 67(5):485-491.
49. Wang JH, Lee CJ, Lee CC, Chen YC, Lee RB and Hsu BG. Fasting Adiponectin is Inversely Correlated with Metabolic Syndrome in Patients with Coronary Artery Disease. *Inter Med* 2010. 49: 739-747.
50. Villarreal-Molina MT, Antuna-Puente B. Adiponectin: Anti-inflammatory and cardioprotective effects. *Biochimie* 2012; 94: 2143-2149.
51. Balsan GA, Vieira JL, Oliveira AM, Portal VL. Relationship between adiponectin, obesity and insulin resistance. *Rev Assoc Med Bras* 2015; 61(1):72-80.
52. Jung CH, Rhee EJ, Choi JH, Bae JC, Yoo SH, Kim WJ and al. The Relationship of Adiponectin/Leptin Ratio with Homeostasis Model Assessment Insulin Resistance Index and Metabolic Syndrome in Apparently Healthy Korean Male Adults. *Korean Diabetes J* 2010; 34: 237-243.
53. Lim S, Quon MJ, Koh KK. Modulation of adiponectin as a potential therapeutic strategy. *Atherosclerosis*. 2014; 233 (2): 721-8.
54. Wang X, Chen Q, Pu H, et al. Adiponectin improves NF- κ B-mediated inflammation and abates atherosclerosis progression in apolipoprotein E-deficient mice. *Lipids in Health and Disease*. 2016; 15:33.
55. Harris MF. The metabolic syndrome. *Aust Fam Physician* 2013; 42: 524-527.

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