



## Body Composition in Healthy Obese/Overweight and Normal Weight Subjects Compared to Patients with Metabolic Syndrome

Somaye Yosae<sup>1</sup>; PhD, Mohammad-Rafi Bazrafshan<sup>2</sup>; PhD, Mohammadreza Erfani<sup>1</sup>; PhD, Alireza Esteghamati<sup>3</sup>; PhD, Banafshe Hosseini<sup>4</sup>; PhD & Kurosh Djafarian<sup>4\*</sup>; PhD

<sup>1</sup> Evaz School of Health, Larestan University of Medical Sciences, Larestan, Iran

<sup>2</sup> Department of Nursing, School of Nursing, Larestan University of Medical Sciences, Larestan, Iran

<sup>3</sup> Endocrinology and Metabolism Research Center (EMRC), Vali-Asr Hospital, Tehran University of Medical Sciences, Tehran, Iran.

<sup>4</sup> Department of Clinical Nutrition, School of Nutritional Sciences and Dietetics, Tehran University of Medical Sciences, Tehran, Iran.

### ARTICLE INFO

#### ORIGINAL ARTICLE

#### Article history:

Received: 20 Apr 2017

Revised: 8 May 2017

Accepted: 13 Sep 2017

#### \*Corresponding author:

kdjafarian@tums.ac.ir  
Department of Clinical  
Nutrition, School of  
Nutritional Sciences and  
Dietetics, Tehran  
University of Medical  
Sciences, Tehran, Iran.

Postal code: 8916188637

Tel: +98 21 88955969

### ABSTRACT

**Background:** According to previous studies, patient with metabolic syndrome (MetS) are different in terms of body composition from healthy subjects. The purpose of the present study was to determine the body composition of healthy obese/overweight patients and compared them with those having MetS. **Methods:** A case-control study was conducted on both men and women aged 20 to 55 years, who were selected using sequential sampling method, based on the inclusion and exclusion criteria, from those referred to an endocrinology and the diabetes clinic affiliated to Tehran University of Medical Sciences. One hundred and forty seven subjects were enrolled in the study and divided into three groups, including 49 with MetS, 49 obese/overweight subjects without MetS, and 49 were normal weight subjects. Body composition was measured for all subjects using bioelectrical impedance analysis. NCEP ATP III was the criterion for definition of MetS. **Results:** No significant differences were found between the study groups in terms of demographic variables. The mean of the waist circumference (WC) was higher in MetS patients ( $P < 0.05$ ) as compared with the control groups. Obese/overweight group had higher percentage of body fat and lower fat free mass than normal weight group ( $P < 0.05$ ). **Conclusion:** Obese/overweight patients with and without MetS had significantly higher fat mass and WC than normal weight controls, while only WC was higher in MetS group as compared with obese/overweight patients without MetS. Therefore, reduction in body fat and WC should be emphasized in patients with MetS.

**Key words:** Metabolic syndrome; Body composition; Obesity.

### Introduction

Metabolic syndrome (MetS), or insulin resistance syndrome, is a combination of coronary heart disease and diabetes mellitus risk

factors including central obesity, hypertension, glucose intolerance, and dyslipidemia (Grundy *et al.*, 2004, Hanson *et al.*, 2002). This definition of

**This paper should be cited as:** Yosae S, Bazrafshan MR, Erfani MR, Esteghamati AR, Hosseini B, Djafarian K. *Body Composition in Healthy Obese/Overweight and Normal Weight Subjects Compared to Patients with Metabolic Syndrome. Journal of Nutrition and Food Security (JNFS)*, 2018; 3 (1): 33-39.

MetS is clinically significant because it can be a strong predictor of cardiovascular diseases and diabetes (Lakka *et al.*, 2002, Wilson *et al.*, 2005). This syndrome is associated with increased risk of diabetes mellitus, stroke, dyslipidemia and coronary heart disease (Jaber *et al.*, 2004, McNeill *et al.*, 2005, Shiwaku *et al.*, 2005). The results of a study conducted by Framingham (Grundy *et al.*, 2004) showed that MetS accounts for about 25% of the new cases of cardiovascular diseases. It has been reported that the prevalence of MetS is at highest rate in Iran and in the world at large. In a study on lipid and glucose, the prevalence of MetS in 42% of women and 24% of men who live in Iran was reported (Azizi *et al.*, 2003).

In both developed and developing countries, the prevalence of obesity has reached epidemic proportions (Ebbeling *et al.*, 2002, Kruger *et al.*, 2006, Veugelers and Fitzgerald, 2005). Obesity and total body fat play key roles in MetS development (Gregor and Hotamisligil, 2011). However, it seems that body fat distribution is more important than amount of body fat. Therefore, body fat distribution may play an important role in the etiology of MetS. Fat accumulation in abnormal area increases the risk of MetS (Alberti *et al.*, 2006, Utzschneider *et al.*, 2004). Visceral fat represents dysfunctional adipose tissue, whose deregulated metabolism, with increased free fatty acid (FFA) flux between the liver and muscle, leads to insulin resistance and worsens dyslipidemia (Avramoglu *et al.*, 2006, Després and Lemieux, 2006).

The prevalence of the MetS is higher in obese patients than in normal weight persons (Goodpaster *et al.*, 2005). However, many obese individuals are not affected by MetS (Stefan *et al.*, 2008, Wildman *et al.*, 2008). In other words, the risk of fat mass threshold for developing MetS is different among individuals. Therefore, the aim of this study was to evaluate the body composition in healthy overweight /obese and normal weight subjects and compared them with patients having MetS.

## Methods and Materials

*Study design and participants:* We conducted a case-control study in Tehran from September 2012 to May 2013. A total of 147 men and women aged between 20 and 55 years participated in this study. The case group consisted of 49 overweight/obese patients with MetS. The matched control group consisted of 49 overweight/obese subjects who were matched to case group for body mass index (BMI), age and gender. Another control group consisted of 49 healthy normal weight subjects who were matched to case group only for age and gender. The BMI cut off for normal weight was 18.5 to 24.9 kg/m<sup>2</sup> and for overweight/obese was BMI = 25 or over 25 kg/m<sup>2</sup>. The cases were recruited from patients referred to the Endocrinology Center of Tehran University of Medical Sciences. The control subjects were selected from those attending the Center for routine medical care. Subjects were selected using sequential sampling method, based on the inclusion and exclusion criteria. The exclusion criteria included having a history of coronary artery disease, acute or chronic renal failure, acute infection within the previous seven days, acute or chronic hepatic failure, hematological disorder, presence of any chronic inflammatory and autoimmune disease, and any known malignancy. Pregnancy, breast feeding, post-menopause, smoking, professional athlete, uncontrolled thyroid disorder, use of medications for dyslipidemia or hypertension, hypnotics, sedatives and immunosuppressive or having a special diet for medical conditions were not included in the study.

*MetS definition:* MetS was defined according to the NCEP ATP III criteria. The ATP III definition requires the presence of three or more of the following: (a) WC equal to or greater than 102 cm for men and greater than or equal to 88 cm for women, (b) triglyceride (TG) level greater than or equal to 150 mg/dL, (c) high-density lipoprotein cholesterol (HDLc) less than 40 mg/dL for men and less than 50 mg/dL for women, (d) systolic blood pressure (SBP) greater than or equal to 135 and/or diastolic blood pressure (DBP) 85 mmHg, and (e) fasting blood glucose (FBG) greater than or

equal to 100 mg/dL.

*Measurements:* Weight was measured by balanced beam scale (Seca Corp. Scale, Germany) with light indoor clothing, and height was measured using standard stadiometer. BMI was calculated as body weight in kg divided by height in meters squared ( $\text{kg}/\text{m}^2$ ). WC was measured by a flexible and non-elastic tape applied in the midline between the lower rib margin and the iliac crest.

Blood samples were collected in the morning, after 8-12 h of overnight fasting and 20 min of supine rest. FBG was measured using an automated glucose oxidase method. TG was measured by glycerol phosphate oxidase, and HDLc was measured after precipitation of the apolipoprotein B containing lipoproteins with phosphotungstic acid (Pars Azmoon Inc., Tehran, Iran).

The fat mass (FM), body fat percentage, and lean body mass (LBM) were measured by means of 8-contact electrode BIA (model TANITA BC-418). This device measures impedance ( $\pm 1 \Omega$ ) and estimates of body composition including %BF ( $\pm 0.1\%$ ), FM ( $\pm 0.1 \text{ kg}$ ), and FFM ( $\pm 0.1 \text{ kg}$ ). For this purpose, individuals were asked to empty their bladder prior to testing and stand on metal footpads in bare feet and grasp a pair of electrodes fixed on a handle. There is a high correlation between the data obtained from BIA and DXA (Ferrari, 2008). Although BIA is less accurate than DXA, it is inexpensive, portable, and relatively simple and fast; thus it measures the body composition objectively with minimal intra- and inter-observer variability (Dehghan and Merchant, 2008).

*Data analysis:* All statistical analyses were performed using SPSS for Windows (version 16.0; SPSS Inc., Chicago, IL, USA). All continuous values were expressed as the mean  $\pm$  SD. Continuous variables were analyzed by one-way ANOVA to compare the difference among the 3 groups. If the result of ANOVA test was

significant, a LSD test was used to determine which mean differs from another. Chi-square and Fisher tests were used to compare the qualitative data. Linear regression was applied to determine the associations between anthropometric indices and selected metabolic syndrome markers in all participants. In all analyses, a two-tailed  $p$ -value  $< 0.05$  was considered statistically significant.

*Ethical considerations:* We obtained informed consent from all the subjects and the study protocol was approved by ethic committee of Tehran University of Medical Sciences.

## Result

The general characteristics of the sample are shown in **Table 1**. Most of the subjects in all groups were men. In terms of age, sex and marital status, there were no statistically significant differences between the three groups. The clinical measures, including blood pressure, TG, SBP and FBG were higher among case patients as compared with both weight matched and non weight matched control subjects (**Table 2**).

The anthropometric and body composition data such as total and regional body composition of subjects for the three groups are shown in **Table 3**. The obese/overweight individuals with and without MetS had a significantly higher total body fat ( $F = 23.08$ ;  $P < 0.001$ ), regional body fat ( $F = 18.8$ ;  $P < 0.001$ ) and BMI ( $F = 83.7$ ;  $P < 0.001$ ) as compared with the normal weight control group, while no significant differences were observed for BF and FFM between the individuals obese/overweight with and without MetS. The mean of FFM percentage was higher in lean control group as compared with the other two groups (**Table 3**). Fat/height ratio ( $F = 35.9$ ;  $P < 0.001$ ) and waist/height ratio ( $F = 66.7$ ;  $P < 0.001$ ) were higher in patient with MetS as compared with control groups (**Table 3**).

**Table 1.** General characteristics of subjects of the study groups

Variables	Case N = 49	Overweight/Obese N = 49	Normal N = 49	Total N = 147	P-value
Sex					0.90 <sup>b</sup>
Male	45 (91.8) <sup>a</sup>	46 (93.9)	45 (91.8)	136 (92.5)	
Female	4 (8.2)	3 (6.1)	4 (8.2)	11 (7.5)	
Marital status					0.39 <sup>b</sup>
Single	8 (16.3)	13 (26.5)	15 (30.6)	36 (24.5)	
Married	41 (83.7)	36 (73.5)	34 (69.4)	111 (75.5)	
Age (year)					0.27 <sup>c</sup>
20-29.9	6 (12.2)	10 (20.4)	15 (30.6)	31 (21.1)	
30-39.9	27 (55.1)	23 (46.9)	24 (49)	74 (50.3)	
40-55	16 (32.7)	16 (32.7)	10 (20.4)	42 (28.6)	

a: N (%), b: Chi-square test, c: Fisher's exact test.

**Table 2.** Comparison of mean ( $\pm$ SD) of biochemistry parameters among subjects of case and control groups

Variables	Case N = 49	Overweight/Obese N = 49	Normal N = 49	Total N = 147	P-value <sup>d</sup>
Fasting blood glucose (mg/dL)	109.0 $\pm$ 48.0 <sup>a</sup>	93.7 $\pm$ 16.9 <sup>b</sup>	91.8 $\pm$ 6.4 <sup>b</sup>	98.2 $\pm$ 30.2	0.008
Triglyceride (mg/dL)	199.8 $\pm$ 95.5 <sup>a</sup>	119.0 $\pm$ 58.5 <sup>b</sup>	109.7 $\pm$ 54.4 <sup>b</sup>	143.0 $\pm$ 82.2	< 0.001
High density lipoprotein cholesterol (mg/dL)	52.2 $\pm$ 7.0 <sup>a</sup>	54.2 $\pm$ 7.0	56.2 $\pm$ 8.0 <sup>b</sup>	54.2 $\pm$ 7.4	0.029
Systolic blood pressure (mmHg)	135.9 $\pm$ 12.76 <sup>a</sup>	127.6 $\pm$ 14.3 <sup>b</sup>	118.9 $\pm$ 12.2 <sup>c</sup>	127.5 $\pm$ 14.7	< 0.001

a, b, c: Dissimilar values of each row are significantly different, d: Values are analyzed by one-way ANOVA.

**Table 3.** Comparison of mean ( $\pm$ SD) total and regional body composition and anthropometric variables among the case and control groups.

Variables	Case N = 49	Overweight/Obese N = 49	Normal N = 49	Total N = 147	P-value <sup>*</sup>
Fat free mass (%)	76.2 $\pm$ 6.09 <sup>a</sup>	76.9 $\pm$ 8.5 <sup>a</sup>	84.1 $\pm$ 6.1 <sup>b</sup>	79.2 $\pm$ 7.8	<0.001
Fat mass (%)	24.7 $\pm$ 8.3 <sup>a</sup>	23.9 $\pm$ 6.0 <sup>a</sup>	15.8 $\pm$ 6.1 <sup>b</sup>	21.4 $\pm$ 7.9	<0.001
Muscle mass (%)	72.7 $\pm$ 5.9 <sup>a</sup>	72.6 $\pm$ 5.8 <sup>a</sup>	80.2 $\pm$ 5.8 <sup>b</sup>	75.3 $\pm$ 6.8	<0.001
Trunk fat (%)	26 $\pm$ 6.1 <sup>a</sup>	26.5 $\pm$ 6.7 <sup>a</sup>	16.4 $\pm$ 6.7 <sup>b</sup>	22/9 $\pm$ 8.0	<0.001
Right hand (%)	23.4 $\pm$ 6.8 <sup>a</sup>	23.4 $\pm$ 6.7 <sup>a</sup>	16.1 $\pm$ 5.3 <sup>b</sup>	20.8 $\pm$ 7.1	0.001
Left hand (%)	23.9 $\pm$ 7.0 <sup>a</sup>	24.5 $\pm$ 7.0 <sup>a</sup>	16.7 $\pm$ 5.7 <sup>b</sup>	21.6 $\pm$ 7.4	<0.001
Right leg (%)	20.6 $\pm$ 6.5 <sup>a</sup>	19.4 $\pm$ 5.7 <sup>a</sup>	14.5 $\pm$ 7.1 <sup>b</sup>	18.1 $\pm$ 6.9	<0.001
Left leg (%)	20.7 $\pm$ 6.4 <sup>a</sup>	19.7 $\pm$ 5.7 <sup>a</sup>	15 $\pm$ 6.6 <sup>b</sup>	18.4 $\pm$ 6.7	<0.001
Height (cm)	172. $\pm$ 6.7 <sup>a</sup>	172.8 $\pm$ 6.9 <sup>a</sup>	172.3 $\pm$ 7.7 <sup>a</sup>	172.4 $\pm$ 7.0	0.862
Weight (kg)	88.7 $\pm$ 11.9 <sup>a</sup>	88.6 $\pm$ 11.4 <sup>a</sup>	68.5 $\pm$ 10.4 <sup>b</sup>	81.7 $\pm$ 14.7	<0.001
Waist circumference (cm)	106.3 $\pm$ 7.47 <sup>a</sup>	102.7 $\pm$ 10.2 <sup>b</sup>	88.3 $\pm$ 6.9 <sup>c</sup>	99.0 $\pm$ 11.3	<0.001
Body mass index (kg/m <sup>2</sup> )	29.9 $\pm$ 3.27 <sup>a</sup>	29.7 $\pm$ 3.21 <sup>a</sup>	22.9 $\pm$ 2.32 <sup>b</sup>	27.5 $\pm$ 4.4	<0.001
Fat/height	0.13 $\pm$ 0.04 <sup>a</sup>	0.12 $\pm$ 0.04 <sup>a</sup>	0.06 $\pm$ 0.02 <sup>b</sup>	0.1 $\pm$ 0.04	<0.001
Waist/height	0.61 $\pm$ 0.04 <sup>a</sup>	0.59 $\pm$ 0.06 <sup>b</sup>	0.51 $\pm$ 0.03 <sup>c</sup>	0.57 $\pm$ 0.06	<0.001

a, b, c: Dissimilar values of each row are significantly different, d: Values are analyzed by one-way ANOVA.

**Table 4.** Correlation between body composition and selected metabolic syndrome markers in all participants (adjusted for gender and age).

Variables	BF (%)		AF (%)		F/H		WC		FFM (%)		MM (%)		BMI	
	$\beta$	R <sup>2</sup>	$\beta$	R <sup>2</sup>	$\beta$	R <sup>2</sup>	$\beta$	R <sup>2</sup>	$\beta$	R <sup>2</sup>	$\beta$	R <sup>2</sup>	$\beta$	R <sup>2</sup>
TG (mg/dL)	1.16	0.06	0.99	0.06	4.45	0.07	1.16	0.07	-1.0	0.06	-1.19	0.06	3.94	0.09
HDLc (mg/dL)	-0.01	0.01	-0.03	0.01	-0.2	0.03	0.10	0.04	0.05	0.02	-0.01	0.01	-0.2	0.03
FBG (mg/dL)	0.39	0.05	0.28	0.05	1.03	0.06	0.35	0.03	0.32	0.05	-0.41	0.05	0.68	0.06
SBP (mmHg)	0.8	0.12	0.72	0.14	2.22	0.16	0.64	0.23	-0.6	0.10	-0.84	0.11	1.55	0.2
DBP (mmHg)	0.66	0.19	0.56	0.19	1.70	0.22	0.46	0.27	-0.6	0.20	-0.73	0.20	1.13	0.25

TG: triglyceride, HDLc: high density lipoprotein cholesterol, FBG: fasting blood glucose, SBP: Systolic blood pressure, DBP: diastolic blood pressure, AF: abdominal fat, WC: waist circumference, FFM: fat free mass, MM: Muscle mass, BMI: body mass index, F/H: fat/height

## Discussion

Previous studies have shown an increased risk of MetS due to change in total and regional body fat (Banerji *et al.*, 1999, Dudeja *et al.*, 2001, Kamath *et al.*, 1999). These documents were extended by comparing the body composition between two groups of obese/overweight with and without MetS, as well as a normal weight group without MetS. To the best of our knowledge, this study is the first to compare the body composition between groups of obese/overweight with and without MetS, as well as a normal weight group without MetS. In this context, the findings of this study showed that although the percentage BF in obese/overweight groups was higher than normal weight group, but there was no significant difference between obese/overweight groups (with and without MetS) in term of total and regional BF. Given that the risk of having MetS will increase in the overweight and obese individuals, there is a considerable variability in the presence of MetS among overweight/obese people. Further researches on the difference between visceral and subcutaneous adipose tissue, as well as molecular factors may yield insights into the mechanisms behind these observations. These results suggest that the differences in body composition cannot serve as a protective

factor against the formation of MetS in the group of obese/overweight patient without MetS, as no significant difference was found for body composition components in these subjects as compared with patients having MetS.

In this study, it was found that WC is a better predictor of MetS than other anthropometric indices. WC was significantly higher in case group as compared with the matched weight control group, as well as normal weight group. WC cannot differentiate the subcutaneous from visceral adipose tissue, but it is strongly correlated with visceral fat and as such, it is a useful indicator for the identification of metabolic disorders (Esmailzadeh *et al.*, 2006, Kullberg *et al.*, 2007, Mukuddem-Petersen *et al.*, 2006, Storti *et al.*, 2006). Furthermore, WC is the best predictor of insulin resistance, the main feature of MetS, as compared with other MetS predictors (Mukuddem-Petersen *et al.*, 2006). According to the findings of Koning *et al.* (De Koning *et al.*, 2007), 1 cm increase in WC is associated with 2% increase in risk of developing cardiovascular disease. Result of other study on Peruvian population showed that the WC and waist-height ratio are the best predictors of MetS (Knowles *et al.*, 2011). The higher WC and waist/height ratio observed in patients with MetS as compared with

obese/overweight subjects without MetS in the present study may partly explain the lower risk of developing MetS in some overweight/obese subjects.

The present study shows that total body fat mass, WC and BMI are positively and significantly associated with SBP and DBP. Also, significant positive associations were observed between BMI and FBS, as well as TG. These results confirmed earlier findings of an association between body composition and hypertension. In addition, the results further support the notion that upper body obesity increases the risk of hypertension and cardiometabolic disorder (Jaddou *et al.*, 2001, Menghetti *et al.*, 2004).

The major limitation of present study was the use of BIA to assess the body composition of subjects and also, the limited number of female participants. Therefore, future studies with similar design and the use of more reliable method to measure the body composition, such as DXA, are needed to confirm our results.

## References

- Alberti KGMM, Zimmet P & Shaw J** 2006. Metabolic syndrome—a new world-wide definition. A consensus statement from the international diabetes federation. *Diabetic medicine*. **23** (5): 469-480.
- Avramoglu RK, Basciano H & Adeli K** 2006. Lipid and lipoprotein dysregulation in insulin resistant states. *International Journal of clinical chemistry and diagnostic laboratory medicine*. **368** (1): 1-19.
- Azizi F, Salehi P, Etemadi A & Zahedi-Asl S** 2003. Prevalence of metabolic syndrome in an urban population: Tehran Lipid and Glucose Study. *Diabetes research and clinical practice*. **61** (1): 29-37.
- Banerji MA, Faridi N, Atluri R, Chaiken RL & Lebovitz HE** 1999. Body composition, visceral fat, leptin, and insulin resistance in Asian Indian men. *The journal of clinical endocrinology & metabolism*. **84** (1): 137-144.
- De Koning L, Merchant AT, Pogue J & Anand SS** 2007. Waist circumference and waist-to-hip ratio as predictors of cardiovascular events: meta-regression analysis of prospective studies. *European heart journal*. **28** (7): 850-856.
- Dehghan M & Merchant AT** 2008. Is bioelectrical impedance accurate for use in large epidemiological studies? *Nutrition journal*. **7** (1): 26.
- Després J-P & Lemieux I** 2006. Abdominal obesity and metabolic syndrome. *Nature*. **444** (7121): 881-887.
- Dudeja V, et al.** 2001. BMI does not accurately predict overweight in Asian Indians in northern India. *British journal of nutrition*. **86** (1): 105-112.
- Ebbeling CB, Pawlak DB & Ludwig DS** 2002. Childhood obesity: public-health crisis, common sense cure. *The lancet*. **360** (9331): 473-482.
- Esmailzadeh A, Mirmiran P & Azizi F** 2006. Comparative evaluation of anthropometric measures to predict cardiovascular risk factors in Tehranian adult women. *Public health nutrition*. **9** (1): 61-69.
- Ferrari CK** 2008. Metabolic syndrome and obesity: Epidemiology and prevention by physical activity and exercise. *Journal of exercise science & fitness*. **6** (2): 87-96.
- Goodpaster BH, et al.** 2005. Obesity, regional body fat distribution, and the metabolic syndrome

## Conclusion

In conclusion, this case-control study provides evidence that obesity itself, independent of its metabolic consequences, is a risk factor for hypertension, increased FBG and TG among obese adults, and introduce low WC as a protective factor against the formation of MetS in obese/overweight without this syndrome.

## Acknowledgments

The authors would like to thank all subjects who took part in the current study.

## Authors' contributions

Yosae S contributed in the conception of the work. Erfani MR and Bazrafshan MR and Hosseini B wrote the manuscript. Esteghamati A and Djafarian k revised the manuscript. All authors read the paper and verified the final version of the manuscript and agreed for all aspects of the work.

## Conflict of interest

There is not conflict of interest.

- in older men and women. *Archives of internal medicine*. **165** (7): 777-783.
- Gregor MF & Hotamisligil GS** 2011. Inflammatory mechanisms in obesity. *Annual review of immunology*. **29**: 415-445.
- Grundy SM, Hansen B, Smith SC, Cleeman JI & Kahn RA** 2004. Clinical management of metabolic syndrome. *Arteriosclerosis, thrombosis, and vascular biology*. **24** (2): e19-e24.
- Hanson RL, Imperatore G, Bennett PH & Knowler WC** 2002. Components of the "metabolic syndrome" and incidence of type 2 diabetes. *Diabetes*. **51** (10): 3120-3127.
- Jaber LA, Brown MB, Hammad A, Zhu Q & Herman WH** 2004. The prevalence of the metabolic syndrome among Arab Americans. *Diabetes care*. **27** (1): 234-238.
- Jaddou HY, Bateiha AM, Khawaldeh A, Goussous YM & Ajlouni KM** 2001. Blood pressure profile in schoolchildren and adolescents in Jordan. *Annals of Saudi medicine*. **21** (1/2): 123-126.
- Kamath SK, et al.** 1999. Cardiovascular disease risk factors in 2 distinct ethnic groups: Indian and Pakistani compared with American premenopausal women. *The American journal of clinical nutrition*. **69** (4): 621-631.
- Knowles K, et al.** 2011. Waist circumference, body mass index, and other measures of adiposity in predicting cardiovascular disease risk factors among Peruvian adults. *International journal of hypertension*. **2011**.
- Kruger R, Kruger H & Macintyre U** 2006. The determinants of overweight and obesity among 10-to 15-year-old schoolchildren in the North West Province, South Africa—the THUSA BANA (Transition and Health during Urbanisation of South Africans; BANA, children) study. *Public health nutrition*. **9** (3): 351-358.
- Kullberg J, et al.** 2007. Practical approach for estimation of subcutaneous and visceral adipose tissue. *Clinical physiology and functional imaging*. **27** (3): 148-153.
- Lakka H-M, et al.** 2002. The metabolic syndrome and total and cardiovascular disease mortality in middle-aged men. *The journal of the American medical association*. **288** (21): 2709-2716.
- McNeill AM, et al.** 2005. The metabolic syndrome and 11-year risk of incident cardiovascular disease in the atherosclerosis risk in communities study. *Diabetes care*. **28** (2): 385-390.
- Menghetti E, et al.** 2004. Hypertension in schoolchildren: research carried out in a secondary school in Rome and observations on dietary patterns. *Minerva pediatrica*. **56** (3): 311-316.
- Mukuddem-Petersen J, et al.** 2006. Sagittal abdominal diameter: no advantage compared with other anthropometric measures as a correlate of components of the metabolic syndrome in elderly from the Hoorn Study. *The American journal of clinical nutrition*. **84** (5): 995-1002.
- Shiwaku K, et al.** 2005. Prevalence of the metabolic syndrome using the modified ATP III definitions for workers in Japan, Korea and Mongolia. *Journal of occupational health*. **47** (2): 126-135.
- Stefan N, et al.** 2008. Identification and characterization of metabolically benign obesity in humans. *Archives of internal medicine*. **168** (15): 1609-1616.
- Storti KL, Brach JS, FitzGerald SJ, Bunker CH & Kriska AM** 2006. Relationships among Body Composition Measures in Community-dwelling Older Women. *Obesity*. **14** (2): 244-251.
- Utzschneider KM, et al.** 2004. Impact of intra-abdominal fat and age on insulin sensitivity and  $\beta$ -cell function. *Diabetes*. **53** (11): 2867-2872.
- Veugelers PJ & Fitzgerald AL** 2005. Prevalence of and risk factors for childhood overweight and obesity. *Canadian medical association journal*. **173** (6): 607-613.
- Wildman RP, et al.** 2008. The obese without cardiometabolic risk factor clustering and the normal weight with cardiometabolic risk factor clustering: prevalence and correlates of 2 phenotypes among the US population (NHANES 1999-2004). *Archives of internal medicine*. **168** (15): 1617-1624.
- Wilson PW, D'Agostino RB, Parise H, Sullivan L & Meigs JB** 2005. Metabolic syndrome as a precursor of cardiovascular disease and type 2 diabetes mellitus. *Circulation*. **112** (20): 3066-3072.