Int J Endocrinol Metab. 2018 April; 16(2):e14159. doi: 10.5812/ijem.14159.

Published online 2018 April 17. **Research Article**

Factor Analysis of Metabolic Syndrome Components in an Iranian Non-Diabetic Adult Population: A Population-Based Study from the North of Iran

Karimollah Hajian-Tilaki[1,*]

[1]Department of Biostatistics and Epidemiology, Babol University of Medical Sciences, Babol, IR Iran

[*]*Corresponding author*: Karimollah Hajian-Tilaki, Department of Biostatistics and Epidemiology, Babol University of Medical Sciences, Babol, IR Iran. E-mail: drhajian@yahoo.com

Received 2017 June 13; **Revised** 2018 January 28; **Accepted** 2018 March 17.

Abstract

Objectives: The aim of this study was to explore the underlying latent factors that can explain the observed variation of components of metabolic syndrome (MetS) in Iranian non-diabetic adult population.

Methods: The researchers performed an exploratory factor analysis (EFA) of metabolic syndrome components, including body mass index (BMI), waist circumference (WC), systolic (SBP) and diastolic blood pressure (DBP), triglyceride (TG), high density lipoprotein (HDL), and Fasting blood sugar (FBS). These observed variables were measured from a representative sample of 841 non-diabetic participants in a cross-sectional population-based study of adults aged 20 to 70 years in the North of Iran.

Results: Three factors were extracted by EFA in both genders. In males, the 3 generated factors were, 1) blood pressure factor underlying systolic and diastolic blood pressure, 2) obesity factor manifested by BMI and WC, 3) lipid/glucose factor underlying TG, HDL and FBS that explained 23.9%, 23.0% and 18.4% of variance in the observed data, respectively, in males. However, in females, BMI and WC were revealed as obesity factors, and systolic and diastolic blood pressure were characterized as hypertension factor, and TG, HDL and FBS appeared to be loaded on lipid/glucose factor, similar to males, and designated 25.6%, 25.4%, and 15.8% of the variance, respectively. Triglyceride and FBS were positively loaded, whereas HDL was loaded negatively with similar loading pattern in both genders. Overall, these 3 underlying latent factors explained 65.3% of the variance of observed clinical data sets in males and 66.8% in females. When TG and HDL were replaced by TG to HDL ratio and also SBP and DBP by mean arterial pressure (MAP), the two-factor model was generated in both genders.

Conclusions: The 2-and 3-factor models were characterized indicating a single pathogenesis that could not explain the unified clustering of MetS in non-diabetic adults.

Keywords: Metabolic Syndrome, Obesity, Dyslipidemia, Insulin Resistance, Blood Pressure, Factor Analysis, Non-Diabetic, Adults

1. Background

Metabolic syndrome (MetS) perceived as a combination of several correlated metabolic disorders appears to increase the risk of occurrence of type 2 diabetes and cardiovascular diseases and their mortality (1). The main concept of its definition is clustering of obesity, hypertension, insulin resistance, and hyperlipidemia. Revean conceptualized its pathophysiologic process in 1988 and called it the "X" syndrome (2). Primarily, its underlying pathophysiologic cause was conceived as insulin resistance (3), yet the possibility of multifactor pathophysiology has been suggested (4). Thus, its definition has been developed over time (5-7). However, the definition of MetS, based on scientific societies may be somewhat different, especially the cut-off points used to define the metabolic abnormality for different components in various regions (5-7).

Nevertheless, there is a consensus definition of components of MetS as clustering of cardio metabolic risk factors. However, previous studies of factor analysis of MetS components yielded inconsistent results (8-11). Thus, the discussion of MetS remains to determine whether a single underlying pathophysiologic exists that unifies the MetS components or it involves a multiple etiologic mechanism. Several models, such as single factor, 2, 3, 4, and even 5 factor models have been suggested in exploratory factor analysis (8, 11, 12). Therefore, the issues of the number of latent factors that can explain the pathophysiologic process remain controversial. Despite an emerging high prevalence of metabolic syndrome in the Iranian population (13-17), especially in the North of Iran (18-21), the data of underlying latent factors are sparse. Therefore, the objective of

this study was to perform exploratory factor analysis to characterize the structure of factors influencing MetS and whether a single pathogenesis plays a central role that unifies the clustering components of MetS in the Iranian non-diabetic adult population.

2. Methods

2.1. Data Sets and Subjects

The data of this analysis were extracted from a population, based on lipid, glucose and metabolic syndrome study that was conducted in Babol, the south of Caspian sea, in the north of Iran, in 2012. A cluster sampling technique with 25 random clusters was used to recruit subjects in a family health survey in the study. The full description of sampling techniques, recruitment criteria, and methods of data collection were explained in details elsewhere (18). The source data included 1000 participants aged 20 to 70 years. For this study, individuals with fasting blood sugar of $\geq$ 126 and/or use of antidiabetic treatment (n = 132) and those with missing data on metabolic variables (n = 7) were excluded. Thus, the data of 841 non-diabetic subjects were entered in this analysis. All subjects had completed a written consent and the study protocol was approved by the Ethics Committee of Babol University of Medical Sciences.

2.2. Measurements of MetS Components

The demographic data, such as age, gender, and prior history of treatment for diabetes and hypertension were collected with an interview. The anthropometric characteristics of weight, height, waist circumference, and hip were measured with standard methods. The systolic and diastolic blood pressure (SBP and DBP) were measured in the sitting position 2 times within a period of 10 minute rest, using a digital sphygmomanometer, following a standardized protocol. The average of these 2 measures was placed in analysis. Additionally, the mean arterial pressure (MAP) was calculated as MAP = DBP + 1/3(SBP-DBP).

All participants were invited to give blood samples after 10 to 12 hours of overnight fasting in the next morning. The fasting blood sugar (FBS), triglyceride (TG), and high density lipoprotein (HDL) cholesterol were measured with a standard enzymatic method in the central lab of Ayatollah Rohani hospital in Babol. For purpose of analysis, TG to HDL ratio (TG/HDL) and also body mass index (BMI) as measures of general adiposity were calculated by weight in kilograms divided by square of height in m2.

2.3. Statistical Analysis

The SPSS software version 18.0 was used for the analysis. The Exploratory principle component (EPC) analysis was performed to determine the underlying latent factors. This factorial analysis was carried out based on 2 sets of observed variables. The first was 7 correlated variables, including SBP, DBP, BMI, WC, TG, HDL, and FBS. Then, TG and HDL were replaced by TG to HDL ratio and also SBP and DBP were replaced by MAP. Thus, this data set included BMI, WC, TG/HDL, MAP, and FBS. Since the distribution of TG/HDL was highly skewed, the log transformation was used in analysis for this variable. For each set of observed variables, EPC analysis was conducted, according to gender. Primarily, the adequacy of samples was tested by the KMO criteria and Bartlett's Chi square test and the significant value of the test showed the adequacy of samples in EPC analysis. The loading factor of each observed variable was estimated and the criteria for including the observed variables in the structure of latent factors was loading of $\geq$ 0.30. The percentage of variance of observed data was calculated, which can be explained by latent factor as a linear combination of observed variables. The communalities of variance of each observed variable that was shared with others in the constructed factor was estimated. The Eigen value (sum of squared loading factor) of $\geq$ 1was used as criteria to include an additional factor in the model. Since the loading coefficients of orthogonal factors are less interpretable, varimax method of rotation was used to extract the most variation of observed data and interpretable observed variables in the structural loading of latent factors.

3. Results

Table 1 shows that the mean age of participants ($\pm$SD) was 41.8 $\pm$ 14.2 and 40.4 $\pm$ 12.3 years (P = 0.12) in males and females, respectively. The males had significantly higher values of WC and systolic, diastolic blood pressure, TG, TG to HDL ratio, and a significant lower level of HDL and BMI, yet FBS levels were not significantly different between genders. The correlation structure between components of Mets is shown in Table 2. Table 3 shows the 3 factors extracted from EFA in males, including: 1) blood pressure factor, 2) obesity factor manifested by BMI and WC, 3) Lipid/glucose factor underlying TG, HDL, and FBS, yet in females obesity (BMI and WC) was characterized as the first factor, and systolic and diastolic blood pressure appeared as the second factor, and lipid/glucose as the third factor. Within the lipid/glucose factor, TG and FBS were positively loaded, whereas HDL was loaded negatively with similar loading pattern in both genders. These 3 underlying latent factors explained 65.3% of variance of observed data sets in

males and 66.8% in females. Table 4 presents the extracted factors and the corresponding loading factors when the data of BMI, WC, MAP, log(TG/HDL), and FBS were used in EFA. The 2-factor model was obtained with rather similar pattern of loading of observed variables between genders. Overall, 57.6% and 61.7% of variation of observed clinical variables were explained by two-factor model in males and females, respectively.

Table 1. Mean of Observed Characteristics of Non-Diabetic Individuals According to Gender[a]

Characteristics	Male (n = 376)	Female (N = 465)	P Value
Age, y	41.8 ± 14.2	40.4 ± 12.3	0.12
BMI, kg/m^2	26.2 ± 4.97	28.1 ± 5.49	0.001
WC, cm	92.5 ± 13.68	90.3 ± 14.65	0.03
DBP, mm/Hg	82.1 ± 13.10	80.4 ± 14.41	0.08
SBP, mm/Hg	127.6 ± 15.41	122.5 ± 18.11	0.001
TG, mg/dL	177.9 ± 128.7	143.5 ± 91.3	0.001
HDL, mg/dL	35.9 ± 9.1	38.8 ± 12.5	0.001
FBS, mg/dL	97.3 ± 11.7	96.7 ± 12.6	0.50
MAP, mm/Hg	97.3 ± 12.73	94.5 ± 14.38	0.003
TG/HDL	5.3 ± 4.59	4.0 ± 3.29	0.001

Abbreviations: BMI, body mass index; DBP, diastolic blood pressure; FBS, fasting blood sugar; HDL, high density lipoprotein; MAP, mean arterial pressure; WC, waist circumference.
[a]Values are expressed as mean ± SD.

4. Discussion

The current results showed that the 2 and 3-factor models could explain the underlying causes of MetS, depending on the dimension of observed variables used in non-diabetic subjects. This evidence does not support the hypothesis of unifying underlying pathologic process; perhaps insulin resistance contributes to MetS and thus ischemic heart diseases, yet shows some measured clinical risk variables associated with more than one factor, indicating the overlap pattern of underlying structure of MetS. This evidence is rather consistent with previous studies on other populations that found at least 2 factors and usually 3 or 4-factor models in both genders (10, 14, 22-28). However, Hanley et al. demonstrated the 2-factor model with similar loading in non-diabetic males and females (10). Similar to the current findings, Sarraf-Zadegn reported a 3 - factor model in Iranian male smokers yet different pattern of loading factors (24). In Chinese adult population with various degrees of insulin sensitivity, the 3-factor model was also suggested (28). Choi et al. found 4 major factors of cardiovascular risk, including impaired glucose tolerance, dyslipidemia, hypertension and obesity, among non-diabetic elderly Korean individuals (22). In another study, through structural equation of metabolic traits, several indicators of abdominal obesity, body mass index, and also lipid and glucose observed variables were entered in the factor analysis; the 5-factor model was extracted and in fact, obesity and some measures of abdominal obesity were extracted as 2 separate factors (23). In contrast, Esteghamati et al. found a single factor model in diabetic and non-diabetic population when TG and HDL were replaced by TG to HDL ratio, as an observed variable (14). In addition, some principal component analyses, using structural equation modeling, reported the fitness of single factor model for MetS components, mentioning the unifying structure of its components (8, 14, 28-30). This inconsistency of findings may be partially attributable to the explanatory nature of PCA and the different extraction methods used to capture all variations of observed variables, not just communal variance that is shared among observed variables (12). Another possible explanation was the number, nature, and dimension of measured variables used in PCA. As the current analysis shows, when the dimension of observed variables was reduced from 7 to 5, by replacing TG and HDL to TG to HDL ratio and calculating MAP, the number of factors was also reduced from 3 to 2 on the same data sets.

The present study shows a minor gender difference in the structure of loading factor by observed variables in the 3-factor model. Hypertension has been shared with relatively stronger association with MetS and greater communalities of variance with other observed variables in males but obesity in females. These results are in accordance with those reported by Shen et al. (9). This reflects that general obesity and abdominal obesity measures are more sensitive indicators of metabolic syndrome for females than males, and hence females are more susceptible to metabolic abnormalities (1, 9, 19). In the current findings, BMI and WC and also TG were loaded significantly on the first factor for females, while in males systolic and diastolic blood pressure had this position. However, the second factor was characterized by BMI, WC, and TG as significant loading in males while blood pressure and FBS had this position in females. The loading pattern of observed variables on the third factor was rather similar between genders, which is an indicator of lipid and glucose profiles. On the other hand, FBS as an indicator of insulin resistance, appeared somewhat more prominent in the second and third factors in females. This may indicate that insulin resistance plays as a central role in the potential impact of MetS (3), and thus MetS is more prevalent in females than males (17, 18). Nonetheless, overall, results showed that the

Table 2. Pearson Correlation Coefficients (P Values) Between the Observed Variables of MetS Components According to Gender

Gender	BMI	WC	DBP	SBP	TG	HDL	FBS
Male							
BMI	1	0.56 (0.001)	0.15 (0.001)	0.21 (0.001)	0.19 (0.001)	-0.006 (NS)	010 (0.02)
WC		1	0.16 (0.001)	0.22 (0.001)	0.16 (0.001)	-0.003 (NS)	0.09 (0.03)
DBP			1	0.66 (0.001)	0.06 (NS)	0.05 (NS)	0.06 (NS)
SBP				1	0.08 (0.04)	0.01 (NS)	0.12 (0.01)
TG					1	-0.17 (0.001)	0.11 (0.02)
HDL						1	-0.16 (0.001)
FBS							1
Female							
BMI	1	0.68 (0.001)	0.21 (0.001)	0.21 (0.001)	0.22 (0.001)	0.008 (NS)	0.09 (0.02)
WC		1	0.30 (0.001)	0.27 (0.001)	0.24 (0.001)	0.06 (NS)	0.08 (0.04)
DBP			1	0.67 (0.001)	0.14 (0.001)	0.08 (0.04)	0.16 (0.001)
SBP				1	0.09 (0.02)	0.11 (0.01)	0.21 (0.001)
TG					1	-0.10	0.12
HLD						1	0.01 (NS)
FBS							1

Abbreviations: BMI, body mass index; DBP, diastolic blood pressure; FBS, fasting blood sugar; HDL, high density lipoprotein; SBP, systolic blood pressure; TG, triglyceride; WC, waist circumference.

Table 3. Three-Factor Model Extracted with the Communalities of Observed Variables and the Loading Factors in Exploratory Factor Analysis Using Orthogonal Rotated Varimax Method with Respect to Gender

Gender/Observed Variables	Communalities	Factor1 Loading Coefficients	Factor2 Loading Coefficients	Factor3 Loading Coefficients
Male[a]				
BMI	0.76	0.10	0.86	0.07
WC	0.75	0.12	0.86	0.04
DBP	0.83	0.91	0.06	-0.005
SBP	0.83	0.89	0.14	0.07
TG	0.39	-0.006	0.30	0.55
HDL	0.61	0.09	0.11	-0.77
FBS	0.41	0.14	0.04	0.62
% of variance	-	23.9	23.0	18.4
% of cumulative variance	-	23.9	46.9	65.3
Female[b]				
BMI	0.80	0.89	0.07	0.06
WC	0.82	0.89	0.17	0.002
DBP	0.75	0.19	0.84	-0.07
SBP	0.79	0.16	0.87	-0.10
TG	0.50	0.37	0.12	0.59
HDL	0.64	0.10	0.15	-0.78
FBS	0.39	0.07	0.50	0.37
% of variance		25.6	25.4	15.8
% of cumulative variance		25.6	51.0	66.8

Abbreviations: BMI, body mass index; WC, waist circumference; DBP, diastolic blood pressure; SBP, systolic blood pressure; TG, triglyceride; HDL. high density lipoprotein; FBS, fasting blood sugar.
[a] Bartlett's test: KMO = 0.57, P = 0.001.
[b] Bartlett's test: KMO = 0.61, P = 0.001.

3-factor model explained a similar observed variability of data, roughly two-thirds in both genders.

Meanwhile, in this study, the two-factor model was extracted by reducing the dimension of observed variables. A more similar pattern of loading factors was found between genders; the first factor was characterized by hypertension/obesity in males, yet hypertension/obesity/lipid in females. Insulin resistance/lipid appeared as a significant loading on the second factor in males and insulin resistance/hypertension also revealed a significant loading on

Table 4. Loading Factors in Exploratory Factor Analysis Using Orthogonal Rotated Varimax Method With Respect to Gender: Two-Factor Model with Reduced Dimension of Observed Data

Gender/Observed Variables	Factor 1 Loading Coefficients	Factor 2 Loading Coefficients
Men[a]		
BMI	0.83	0.11
WC	0.84	0.09
MAP	0.50	0.02
TG/HDL	0.11	0.72
FBS	0.02	0.78
% of variance	33.3	22.9
% of cumulative variance	33.3	56.2
Women[b]		
BMI	0.88	0.04
WC	0.90	0.08
MAP	0.36	0.57
TG/HDL	0.34	0.19
FBS	-0.04	0.89
% of variance	33.3	22.9
% of cumulative variance	33.3	56.2

Abbreviations: BMI, body mass index; FBS, fasting blood sugar; HDL, high density lipoprotein; KMO, Kaiser-Meyer-Olkin Measure of sampling adequacy; MAP, mean arterial pressure; TG, triglyceride; WC, waist circumference.
[a] Bartlett's test: KMO = 0.61, P = 0.001.
[b] Bartlett's test: KMO = 0.61, P = 0.001.

the second factor in females. In contrast to the study of Hanley et al., the two-factor model had a similar pattern of loading of observed variables between genders, where obesity/lipid/glucose profiles were revealed significantly on the first factor and hypertension on the second factor (10).

This study may have some limitations. The cross-sectional condition of this study precludes any interpretation of findings in terms of causality. It is conceivable that the risk variables influence each other in reciprocal direction. In addition, this study only evaluated the association of traditional risk factors of MetS and did not measure homeostasis model assessment (HOMA) as a measure of insulin resistance and other nontraditional risk factors, such as inflammatory-related factors. However, the strength of this study was being population-based with objective inclusion and exclusion criteria to recruit non-diabetic individuals in analysis, using standard methods of sample selection and standard measurement of data collection. For future studies, this will definitely contribute to the understanding of pathogenesis of MetS if prospective design is established and the pathway causal relationship is assessed with a clear temporal sequence.

5. Conclusions

The 2-and the 3-factor models were identified and none of the observed variables loaded on all factors indicated that more than one pathophysiologic mechanism is plausible to contribute for the clustering of metabolic risk factors in non-diabetic adults.

Acknowledgments

The authors would like to thank the deputy for research and technology of Babol University of Medical Sciences.

Footnote

Conflict of Interest: The author declares that there was no conflict of interest to disclose.

References

1. Ford ES. The metabolic syndrome and mortality from cardiovascular disease and all-causes: findings from the National Health and Nutrition Examination Survey II Mortality Study. *Atherosclerosis*. 2004;**173**(2):309–14. doi: 10.1016/j.atherosclerosis.2003.12.022. [PubMed: 15064107].
2. Reaven GM. Banting Lecture 1988. Role of insulin resistance in human disease. 1988. *Nutrition*. 1997;**13**(1):65. discussion 64, 66. [PubMed: 9058458].
3. Reaven G. Insulin resistance, type 2 diabetes mellitus, and cardiovascular disease: the end of the beginning. *Circulation*. 2005;**112**:30–2.
4. Kahn R, Buse J, Ferrannini E, Stern M, American Diabetes A, European Association for the Study of D. The metabolic syndrome: time for a critical appraisal: joint statement from the American Diabetes Association and the European Association for the Study of Diabetes. *Diabetes Care*. 2005;**28**(9):2289–304. [PubMed: 16123508].
5. Expert Panel on Detection E, Treatment of High Blood Cholesterol in A. 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**(19):2486–97. [PubMed: 11368702].
6. Alberti KG, Zimmet P, Shaw J, I. D. F. Epidemiology Task Force Consensus Group . The metabolic syndrome–a new worldwide definition. *Lancet*. 2005;**366**(9491):1059–62. doi: 10.1016/S0140-6736(05)67402-8. [PubMed: 16182882].
7. Grundy SM, Brewer HJ, Cleeman JI, Smith SJ, Lenfant C, American Heart A, et al. Definition of metabolic syndrome: Report of the National Heart, Lung, and Blood Institute/American Heart Association conference on scientific issues related to definition. *Circulation*. 2004;**109**(3):433–8. doi: 10.1161/01.CIR.0000111245.75752.C6. [PubMed: 14744958].

8. Shen BJ, Todaro JF, Niaura R, McCaffery JM, Zhang J, Spiro A3, et al. Are metabolic risk factors one unified syndrome? Modeling the structure of the metabolic syndrome X. *Am J Epidemiol*. 2003;**157**(8):701–11. [PubMed: 12697574].
9. Shen BJ, Goldberg RB, Llabre MM, Schneiderman N. Is the factor structure of the metabolic syndrome comparable between men and women and across three ethnic groups: the Miami Community Health Study. *Ann Epidemiol*. 2006;**16**(2):131–7. doi: 10.1016/j.annepidem.2005.06.049. [PubMed: 16257230].
10. Hanley AJ, Karter AJ, Festa A, D'Agostino RJ, Wagenknecht LE, Savage P, et al. Factor analysis of metabolic syndrome using directly measured insulin sensitivity: The Insulin Resistance Atherosclerosis Study. *Diabetes*. 2002;**51**(8):2642–7. [PubMed: 12145182].
11. Novak S, Stapleton LM, Litaker JR, Lawson KA. A confirmatory factor analysis evaluation of the coronary heart disease risk factors of metabolic syndrome with emphasis on the insulin resistance factor. *Diabetes Obes Metab*. 2003;**5**(6):388–96. [PubMed: 14617224].
12. Shah S, Novak S, Stapleton LM. Evaluation and comparison of models of metabolic syndrome using confirmatory factor analysis. *Eur J Epidemiol*. 2006;**21**(5):343–9. doi: 10.1007/s10654-006-9004-2. [PubMed: 16736276].
13. Fakhrzadeh H, Ebrahimpour P, Pourebrahim R, Heshmat R, Larijani B. Metabolic Syndrome and its Associated Risk Factors in Healthy Adults: APopulation-Based Study in Iran. *Metab Syndr Relat Disord*. 2006;**4**(1):28–34. doi: 10.1089/met.2006.4.28. [PubMed: 18370767].
14. Esteghamati A, Zandieh A, Khalilzadeh O, Meysamie A, Ashraf H. Clustering of metabolic syndrome components in a Middle Eastern diabetic and non-diabetic population. *Diabetol Metab Syndr*. 2010;**2**:36. doi: 10.1186/1758-5996-2-36. [PubMed: 20529329].
15. Azizi F, Salehi P, Etemadi A, Zahedi-Asl S. Prevalence of metabolic syndrome in an urban population: Tehran Lipid and Glucose Study. *Diabetes Res Clin Pract*. 2003;**61**(1):29–37. [PubMed: 12849921].
16. Sarrafzadegan N, Kelishadi R, Baghaei A, Hussein Sadri G, Malekafzali H, Mohammadifard N, et al. Metabolic syndrome: an emerging public health problem in Iranian women: Isfahan Healthy Heart Program. *Int J Cardiol*. 2008;**131**(1):90–6. doi: 10.1016/j.ijcard.2007.10.049. [PubMed: 18190978].
17. Hajian-Tilaki K. Metabolic syndrome and its associated risk factors in Iranian adults: A systematic review. *Caspian J Intern Med*. 2015;**6**(2):51–61. [PubMed: 26221500].
18. Hajian-Tilaki K, Heidari Firozjahi B. Prevalence of metabolic syndrome and the associated socio-demographic characteristics and physical activity in urban population of Iranian adults:. *Diabetes Metab Synd Clin Rev*. 2014;**8**:170–6.
19. Hajian-Tilaki KO, Heidari B. Prevalence of obesity, central obesity and the associated factors in urban population aged 20-70 years, in the north of Iran: a population-based study and regression approach. *Obes Rev*. 2007;**8**(1):3–10. doi: 10.1111/j.1467-789X.2006.00235.x. [PubMed: 17212790].
20. Hajian-Tilaki K, Heidari B. Is waist circumference a better predictor of diabetes than body mass index or waist-to-height ratio in Iranian adults? *Int J Prev Med*. 2015;**6**:5. doi: 10.4103/2008-7802.151434. [PubMed: 25789140].
21. Hajian-Tilaki K, Heidari B, Hajian-Tilaki A, Firouzjahi A, Bagherzadeh M. The discriminatory performance of body mass index, waist circumference, waist-to-hip ratio and waist-to-height ratio for detection of metabolic syndrome and their optimal cutoffs among Iranian adults. *J Res Health Sci*. 2014;**14**(4):276–81. [PubMed: 25503283].
22. Choi KM, Lee J, Kim KB, Kim DR, Kim SK, Shin DH, et al. Factor analysis of the metabolic syndrome among elderly Koreans–the South-west Seoul Study. *Diabet Med*. 2003;**20**(2):99–104. [PubMed: 12581260].
23. Karns R, Succop P, Zhang G, Sun G, Indugula SR, Havas-Augustin D, et al. Modeling metabolic syndrome through structural equations of metabolic traits, comorbid diseases, and GWAS variants. *Obesity*. 2013;**21**(12).
24. Sarraf-Zadegan N, Baghaei AM, Sadeghi M, Amin-Zadeh A. Factor analysis of metabolic syndrome among Iranian male smokers. *Iran J Med Sci*. 2015;**30**(2).
25. Nasila Sungwacha J, Tyler J, Longo-Mbenza B, Lasi On'Kin JB, Gombet T, Erasmus RT. Assessing clustering of metabolic syndrome components available at primary care for Bantu Africans using factor analysis in the general population. *BMC Res Notes*. 2013;**6**:228. doi: 10.1186/1756-0500-6-228. [PubMed: 23758878].
26. Ayubi E, Khalili D, Delpisheh A, Hadaegh F, Azizi F. Factor analysis of metabolic syndrome components and predicting type 2 diabetes: Results of 10-year follow-up in a Middle Eastern population. *J Diabetes*. 2015;**7**(6):830–8. doi: 10.1111/1753-0407.12252. [PubMed: 25492310].
27. Oh JY, Hong YS, Sung YA, Barrett-Connor E. Prevalence and factor analysis of metabolic syndrome in an urban Korean population. *Diabetes Care*. 2004;**27**(8):2027–32. [PubMed: 15277435].
28. Anderson PJ, Critchley JA, Chan JC, Cockram CS, Lee ZS, Thomas GN, et al. Factor analysis of the metabolic syndrome: obesity vs insulin resistance as the central abnormality. *Int J Obes Relat Metab Disord*. 2001;**25**(12):1782–8. doi: 10.1038/sj.ijo.0801837. [PubMed: 11781758].
29. Pladevall M, Singal B, Williams LK, Brotons C, Guyer H, Sadurni J, et al. A single factor underlies the metabolic syndrome: a confirmatory factor analysis. *Diabetes Care*. 2006;**29**(1):113–22. [PubMed: 16373906].
30. Huo D, Wang W, Li X, Gao Q, Wu L, Luo Y, et al. Evaluation of two single-factor models of metabolic syndrome: a confirmatory factor analysis for an adult population in Beijing. *Lipids Health Dis*. 2013;**12**:61. doi: 10.1186/1476-511X-12-61. [PubMed: 23638905].