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

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 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.

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. Reave 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).

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, 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 ≥ 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 Kohani 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 ≥ 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 ≥ 1 was 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 (±SD) was 41.8 ± 14.2 and 40.4 ± 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.

| Characteristics | Male (n = 376) | Female (N = 465) | P Value |
|-----------------|---------------|-----------------|--------|
| Age, y          | 41.8 ± 14.2   | 40.4 ± 12.3     | 0.32   |
| BMI, kg/m²      | 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      | 127.0 ± 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  |

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-Zadeh 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, Eshteghamati 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 3-factor model explained a similar observed variability of data, roughly two-thirds in both genders.
### 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) | 0.20 (0.001) | 0.19 (0.001) | -0.006 (NS) | 0.10 (0.02) |
| WC     | 1   | 0.66 (0.001) | 0.16 (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)   | 0.06 (NS)   |
| SBP    | 1   | 0.00 (NS)    | 0.01 (NS)    | 0.01 (NS)    | 0.01 (NS)   | 0.01 (NS)   |
| TG     | 1   | -0.17 (0.001) | 0.11 (0.02)  | -0.16 (0.001) | 0.01 (NS)   | 0.01 (NS)   |
| HDL    | 1   | 0.00 (NS)    | 0.00 (NS)    | 0.00 (NS)    | 0.00 (NS)   | 0.00 (NS)   |
| FBS    | 1   | -0.00 (NS)   | -0.00 (NS)   | -0.00 (NS)   | -0.00 (NS)  | -0.00 (NS)  |
| Female |     |    |     |     |    |     |     |
| BMI    | 1   | 0.68 (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) | 0.06 (NS)   |
| DBP    | 1   | 0.67 (0.001) | 0.14 (0.001) | 0.08 (NS)    | 0.16 (0.001) |
| SBP    | 1   | 0.09 (0.04)  | 0.11 (0.01)  | 0.21 (0.001) |
| TG     | 1   | -0.10        | 0.12         | -0.10        |
| HDL    | 1   | 0.01 (NS)    | 0.01 (NS)    | 0.01 (NS)    |
| FBS    | 1   | 0.00 (NS)    | 0.00 (NS)    | 0.00 (NS)    |

### 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 | Factor 1 Loading Coefficients | Factor 2 Loading Coefficients | Factor 3 Loading Coefficients |
|---------------------------|---------------|-------------------------------|-------------------------------|-------------------------------|
| Male                      |               |                               |                               |                               |
| BMI                       | 0.76          | 0.10                          | 0.86                          | 0.07                          |
| WC                        | 0.75          | 0.12                          | 0.86                          | 0.04                          |
| DBP                       | 0.43          | 0.09                          | 0.84                          | -0.003                       |
| SBP                       | 0.43          | 0.49                          | 0.04                          | 0.07                          |
| TG                        | 0.39          | 0.004                         | 0.30                          | 0.55                          |
| HDL                       | 0.41          | 0.19                          | 0.16                          | -0.77                         |
| FBS                       | 0.41          | 0.04                          | 0.04                          | 0.62                          |
| % of variance             |               | 21.9                          | 21.0                          | 18.8                          |
| % of cumulative variance  |               | 23.9                          | 46.5                          | 55.1                          |
| Female                    |               |                               |                               |                               |
| BMI                       | 0.80          | 0.07                          | 0.06                          |                               |
| WC                        | 0.82          | 0.09                          | 0.07                          | 0.06                          |
| DBP                       | 0.75          | 0.13                          | 0.84                          | -0.07                         |
| SBP                       | 0.75          | 0.16                          | 0.87                          | -0.10                         |
| TG                        | 0.50          | 0.37                          | 0.12                          | 0.59                          |
| HDL                       | 0.50          | 0.40                          | 0.05                          | -0.78                         |
| FBS                       | 0.39          | 0.07                          | 0.50                          | 0.37                          |
| % of variance             |               | 25.4                          | 25.4                          | 15.8                          |
| % of cumulative variance  |               | 25.4                          | 51.0                          | 66.8                          |

| Bartlett’s test: KMO = 0.57, P = 0.001. |
| Bartlett’s test: KMO = 0.61, P = 0.001. |

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 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 correction. 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 model were identified and none of the observed variables loaded on all factors indicated that more than one pathophysiological mechanism is plausible to contribute for the clustering of metabolic risk factors in non-diabetic adults.

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Footnote

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

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