Association of Z-Score of the Log-Transformed A Body Shape Index with Cardiovascular Disease in People Who Are Obese but Metabolically Healthy: The Korea National Health and Nutrition Examination Survey 2007–2010

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Background: We aimed at evaluating the effect of the z-score of the log-transformed A Body Shape Index (LB-SIZ) on cardiovascular disease (CVD) outcomes according to obesity phenotype.

Methods: Data were collected from the Korea National Health and Nutrition Examination Survey conducted from 2007 to 2010. Obesity was defined as a body mass index above 25 kg/m² and metabolic abnormality was defined as the presence of two or more metabolic risk factors of the Adult Treatment Panel III definition. The participants were classified by obesity and metabolic healthy status: metabolically healthy non-obese (MHNO), metabolically healthy obese (MHO), metabolically unhealthy non-obese (MUNO), and metabolically unhealthy obese (MUO). Each group was further classified into three groups based on the tertile of LB-SIZ. A multivariate logistic regression analysis with adjustment for age, sex, smoking status, income, education level, physical activities, alcohol, and energy intake was conducted to evaluate the odds ratio (OR) for CVD events.

Results: In the multivariate logistic regression model, MHO participants who are within the third tertile of LB-SIZ had a significantly higher OR for CVD events, whereas those who are within the first and second tertile of LB-SIZ were not at high risk of developing CVDs compared to MHNO participants who are within the first tertile of LB-SIZ. In addition, a similar increase in the OR was observed in MUNO or MUO participants.

Conclusion: LB-SIZ had the lowest risk for CVDs in the first tertile of LB-SIZ and a linear relationship with all its tertiles in MHO, MUNO, and MUO participants.

Key words: Obesity, Metabolically benign, Body mass index, Body constitution, Cardiovascular disease

INTRODUCTION

The World Health Organization (WHO) reported that 13% of the world’s adult population were obese (body mass index [BMI] ≥ 30 kg/m²) in 2016 and the worldwide prevalence of obesity was nearly tripled between 1975 and 2016.¹ According to the Korea National Health and Nutrition Examination Survey (KNHANES), the prevalence of obesity with a BMI ≥ 25 kg/m² in Korean adults increased from 25.8% in 1998 to 31.5% in 2014.² This increase is particularly significant when the association between obesity and poor clinical outcomes, such as type 2 diabetes mellitus (T2DM), cardiovascular diseases (CVDs), stroke, and mortality, is considered.³⁴ However, obese individuals do not always have metabolic dysfunction. This type of obesity is referred to as metabolically healthy obesity phenotype. Although, by definition, individuals with metabolically healthy obesity are metabolically healthy, the...
clinical outcomes of this condition remain controversial. Hamer and Stamatakis' have conducted a 7-year observational study involving 22,203 participants without CVD, and failed to show the association between metabolically healthy obesity and the risk for CVD or all-cause mortality. In addition, several observational studies have shown that metabolically healthy obesity did not increase the risk for coronary atherosclerosis and heart failure. On the other hand, several large-scale population-based studies have reported that metabolically healthy obesity is considered as a significant risk factor for CVDs. The differences in these results suggest that metabolically healthy obesity phenotype represents a heterogeneous group. Therefore, the key factors that determine poor clinical outcomes in individuals with metabolically healthy obesity must be investigated.

In the present study, we focused on the drawback of BMI as a key factor for the heterogeneous prognosis. BMI cannot distinguish between muscle and fat, and it cannot accurately predict the percentage of body fat. In addition, several epidemiological studies have reported the limitations of BMI in predicting the risk of heart attack, stroke, and death. To overcome this, Krakauer and Krakauer developed A Body Shape Index (ABSI), a new equations for estimating body shape using waist circumference (WC), weight, and height in the U.S. population. However, there have been few studies to examine the usefulness of ABSI in the other ethnic group. Therefore, we used the z-score of the log-transformed A Body Shape Index (LBSIZ) to control for age, sex, and ethnic differences. This study aimed at assessing the effect of body shape index on CVD outcomes according to obesity phenotype in a representative sample of the Korean population.

**METHODS**

**Study population**

Data were collected from the KNHANES conducted from 2007 to 2010. The surveys were cross-sectional and nationally representative with a multistage and stratified sample design. In total, 33,829 participants were included in the study. Participants with missing data or those under 20 years of age were excluded. The total number of eligible participants was 21,948 (Fig. 1).

![Flowchart for final selection](https://orcid.org/0000-0003-3298-3630)

### Clinical and laboratory measurements

WC was measured at the midpoint between the lowest border of the rib cage and the upper lateral border of the iliac crest at the end of normal expiration. Blood pressure (BP) was measured three times while the patient is in a sitting position after at least 5 minutes of rest. An average of three recorded BP measurements was used. After an 8-hour overnight fasting, triglyceride (TG), high-density lipoprotein cholesterol (HDL-C), and glucose levels were measured using ADVIA 1650 (Siemens, Washington, DC, USA) in 2007 and Hitachi Automatic Analyzer 7600 (Hitachi, Tokyo, Japan) from 2008 to 2010. We used the revised HDL-C values between 2007 and 2010 according to the Korea Centers for Disease Control and Prevention (KCDC) guidelines for consistency in each survey.

**Measurement of obesity**

In the present study, obesity was defined as a BMI above 25 kg/m² based on the Asia-Pacific BMI criteria by the WHO Western Pacific Region. Because of the lack of a standard definition for metabolic health and obesity, we used the National Cholesterol Education Program—Adult Treatment Panel III (NCEP–ATP III) criteria for metabolic syndrome, which is the most frequently used definition in recent studies. Briefly, metabolic health was defined as ≤ 1 of the following NCEP–ATP III criteria, including impaired fasting glucose level (at least 100 mg/dL), hypertension (a systolic BP above 130 mmHg and/or a diastolic BP above 85 mmHg or the use of antihypertensive drugs), TG level of at least 150 mg/dL, and a low level of HDL-C (40 mg/dL in men and 50 mg/dL in women). Due to the collinearity between BMI and WC, the central obesity criterion was not used. The participants were classified based on these criteria: metabolically healthy non-obese (MHNO),
metabolically healthy obese (MHO), metabolically unhealthy non-obese (MUNO), and metabolically unhealthy obese (MUO).

ABSI is calculated using the following equation:\(^\text{16}\):

\[
\text{ABSI} = \frac{\text{WC} \times \text{weight}^{-2/3} \times \text{height}^{5/6}}{\text{BMI}^{2/3} \times \text{height}^{1/2}}
\]

However, because of its statistical limitations in terms of skewness and symmetry, the z-score of the log-transformed ABSI was used. The detailed equation used for the calculation of LBSIZ was described in our previous study.\(^\text{17}\)

Statistical analysis
Summary statistics are presented as mean and standard deviation or prevalence (%). One-way analysis of variance and the Pearson chi-square test were used to compare each variable according to obesity phenotypes. A multivariate logistic regression analysis with adjustment for age, sex, smoking status, income, education level, physical activities, alcohol, and energy intake was carried out to evaluate the odds ratio (OR) and 95% confidence interval (CI) for CVD events according to obesity phenotype and LBSIZ. The graphical relationships were also evaluated with restricted cubic spline plots with three knots according to obesity phenotypes. Analyses were performed using IBM SPSS version 24.0 (IBM Corp., Armonk, NY, USA) and the statistical program R (R version 3.1.0, 2014; https://www.r-project.org). A P-value of 0.05 was considered statistically significant.

Ethical considerations
The study protocol was approved by the Institutional Review Board of the KCDC (2007-02CON-04-P, 2008-04EXP-01-C, 2009-01CON-03-2C, 2010-02CON21-C). Written informed consent was obtained from each study participant prior to the survey.

RESULTS
Baseline characteristics of the participants
The data of 21,948 participants were assessed. The general characteristics of the participants according to obesity phenotypes are summarized in Table 1.

| Variable                  | MHNO (n = 10,134) | MHO (n = 2,665) | MUNO (n = 4,822) | MUO (n = 4,327) | P   |
|---------------------------|-------------------|-----------------|------------------|-----------------|-----|
| Male sex                  | 3,766 (37.2)      | 1,235 (46.3)    | 2,301 (47.7)     | 2,093 (48.4)    | <0.001 |
| Age (yr)                  | 43.7 ± 15.4       | 45.7 ± 14.6     | 57.8 ± 14.7      | 54.4 ± 14.2     | <0.001 |
| CVD events*               | 193 (1.9)         | 78 (2.9)        | 334 (6.9)        | 278 (6.4)       | <0.001 |
| Smoking                   | 3,801 (37.5)      | 1,144 (42.9)    | 2,235 (46.4)     | 1,967 (45.5)    | <0.001 |
| BMI (kg/m\(^2\))         | 21.6 ± 2.1        | 27.1 ± 2.0      | 22.6 ± 1.8       | 27.6 ± 2.4      | <0.001 |
| Waist circumference (cm)  | 75.2 ± 7.2        | 88.0 ± 7.1      | 80.8 ± 6.9       | 92.0 ± 7.0      | <0.001 |
| LBSIZ                     | –0.2 ± 1.0        | –0.2 ± 1.0      | 0.4 ± 1.0        | 0.1 ± 0.9       | <0.001 |
| Systolic BP (mmHg)        | 109.6 ± 13.9      | 115.3 ± 14.1    | 126.1 ± 18.2     | 127.1 ± 16.6    | <0.001 |
| Diastolic BP (mmHg)       | 71.1 ± 9.2        | 75.2 ± 9.2      | 78.1 ± 10.8      | 80.8 ± 10.4     | <0.001 |
| FBG level (mg/dL)         | 88.8 ± 11.9       | 92.7 ± 13.5     | 106.6 ± 30.8     | 109.0 ± 29.2    | <0.001 |
| HbA1c (%)                 | 7.1 ± 1.8         | 7.0 ± 1.3       | 7.4 ± 1.5        | 7.3 ± 1.4       | 0.120 |
| Total cholesterol (mg/dL) | 180.0 ± 32.8      | 183.7 ± 35.7    | 192.7 ± 38.1     | 198.0 ± 37.6    | <0.001 |
| HDL-C (mg/dL)             | 52.7 ± 10.4       | 49.6 ± 9.4      | 42.9 ± 9.2       | 41.7 ± 8.4      | <0.001 |
| TG (mg/dL)                | 87.8 ± 45.2       | 107.3 ± 57.8    | 184.8 ± 131.1    | 201.8 ± 135.5   | <0.001 |
| Metabolic state           |                   |                 |                  |                 |     |
| Central obesity           | 1,252 (12.4)      | 1,894 (71.1)    | 1,403 (29.1)     | 3,544 (81.9)    | <0.001 |
| High BP                   | 1,210 (11.9)      | 520 (19.8)      | 3,110 (64.5)     | 3,037 (70.2)    | <0.001 |
| Hyperglycemia             | 710 (7.0)         | 268 (10.1)      | 2,520 (52.3)     | 2,482 (57.4)    | <0.001 |
| Low HDL-C level           | 2,607 (25.7)      | 755 (28.3)      | 3,484 (72.3)     | 3,170 (73.3)    | <0.001 |
| High TG level             | 521 (5.1)         | 247 (9.3)       | 2,783 (57.7)     | 2,800 (64.7)    | <0.001 |

Values are presented as number (%) or mean ± standard deviation. *Participants who had either myocardial infarction, coronary heart disease, congestive heart failure, cerebrovascular disease, or peripheral arterial disease. MHNO, metabolically healthy non-obese; MHO, metabolically healthy obese; MUNO, metabolically unhealthy non-obese; MUO, metabolically unhealthy obese; CVD, cardiovascular disease; BMI, body mass index; LBSIZ, z-score of the log-transformed A Body Shape Index; BP, blood pressure; FBG, fasting blood glucose; HbA1c, hemoglobin A1c; HDL-C, high-density lipoprotein cholesterol; TG, triglyceride.
Association between obesity phenotypes and risk of CVD events

A total of 883 CVD events were reported. In the multivariate logistic regression model, participants with MHO were at moderate risk of developing CVD compared with participants with MHNO (OR, 1.485; 95% CI, 1.031–2.139), whereas participants with metabolic abnormalities were at high risk regardless of obesity (MUNO: OR, 1.768; 95% CI, 1.373–2.278; MUO: OR, 2.179; 95% CI, 1.664–2.855) (Table 2). Although the CVD events were most frequently reported in MUNO group, MUO showed the highest OR for CVD event after adjustment for the confounding factors.

Risk of developing CVDs according to obesity phenotypes and LBSIZ

We divided the LBSIZ into tertiles and examined the relative risk of developing CVDs across the tertiles based on obesity phenotype. The distribution of LBSIZ was described in Table 3 according to obesity phenotype. In the multivariate logistic regression analysis, MHO participants who are within the third tertiles of LBSIZ had a significantly higher OR for CVDs, whereas those who are within the first and second tertile of LBSIZ were not at risk for CVD compared to MHNO participants who are within the first tertile. In addition, the ORs of the MUNO and MUO participants increased similarly (Fig. 2). Because age and sex ratio were significantly different across the subgroups by obesity phenotypes, further analysis with propensity score matching methods was performed and showed similar results (Table 4). In the restricted cubic spline regression, LBSIZ showed a linear relationship with CVD events according to each obesity phenotype (Fig. 3).

**DISCUSSION**

In this study, we investigated the risk for CVD events based on MHO phenotype. MHO participants are at moderate risk for CVD than MHNO participants. Notably, we found that LBSIZ had a positive linear relationship with CVD events in MHO participants. Although, the linear relationship disappeared in MUO participants after weighting, similar pattern was shown in MHNO and MUO participants in the unweighted logistic regression analysis and the restricted cubic spline regression.

These results show that patients with MHO phenotype are not homogenous. This heterogeneity might be due to a limitation in the definition of obesity based on BMI, which is used to define the severity of obesity in the general population based on the guidelines of the WHO.20 However, BMI could not accurately obtain body fat mass because body weight cannot be used to differentiate excess fat from bone or muscle mass.28 In addition, BMI does not consider age-related changes in muscle mass.29 Although body fat increases and muscle mass decreases with age, BMI may not reflect the proportional changes in body fat or muscle mass.29,30 Other disadvantages of using BMI are the following: BMI cannot not measure the regional body fat composition, and it does not consider the distribution of fats in terms of sex. In addition, the metabolic

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### Table 2. Odds ratios for CVD events according to obesity phenotypes

| Variable  | Unweighted model | Weighted model |
|-----------|------------------|----------------|
|           | Odds ratio (95% CI) | P | Odds ratio (95% CI) | P |
| MHNO      | 1 (Reference)     |    | 1 (Reference)      |    |
| MHO       | 1.568 (1.194–2.060) | 0.001 | 1.485 (1.031–2.139) | 0.034 |
| MUNO      | 1.758 (1.456–2.122) | < 0.001 | 1.768 (1.373–2.278) | < 0.001 |
| MUO       | 2.147 (1.770–2.603) | < 0.001 | 2.179 (1.664–2.855) | < 0.001 |

Adjusted for age, sex, family income level, education level, moderate intensity physical activities (> 120 minute per week), alcohol consumption, smoking status, and energy intake.

CVD, cardiovascular disease; CI, confidence interval; MHNO, metabolically healthy non-obese; MHO, metabolically healthy obese; MUNO, metabolically unhealthy non-obese; MUO, metabolically unhealthy obese.

### Table 3. The distribution of LBSIZ by obesity phenotype

| Percentile  | 10th | 20th | 30th | 40th | 50th | 60th | 70th | 80th | 90th |
|-------------|------|------|------|------|------|------|------|------|------|
| Total       | −1.28 | −0.84 | −0.53 | −0.25 | 0    | 0.25 | 0.50 | 0.81 | 1.24 |
| MHNO        | −1.43 | −1.02 | −0.70 | −0.44 | −0.19 | 0.06 | 0.32 | 0.60 | 1.01 |
| MHO         | −1.47 | −0.99 | −0.71 | −0.46 | −0.21 | 0.04 | 0.28 | 0.57 | 1.00 |
| MUNO        | −0.90 | −0.46 | −0.15 | 0.13  | 0.37 | 0.62 | 0.88 | 1.19 | 1.59 |
| MUO         | −1.08 | −0.64 | −0.32 | −0.08 | 0.14 | 0.37 | 0.59 | 0.88 | 1.30 |

LBSIZ, z-score of the log-transformed A Body Shape Index; MHNO, metabolically healthy non-obese; MHO, metabolically healthy obese; MUNO, metabolically unhealthy non-obese; MUO, metabolically unhealthy obese.
effects of fat differ according to its location. For instance, visceral fat is associated with T2DM and CVDs, whereas subcutaneous fat has beneficial effects on metabolism. Therefore, BMI has limitations in terms of classifying obesity. A person with excessive visceral fat and insufficient muscle mass can have a normal BMI but have a high mortality risk.

A number of studies attempted to investigate obesity using other body index parameters, such as WC, because of the limitations of BMI. WC is strongly associated with visceral adiposity and metabolic risk and increased morbidity and mortality. However, WC could not distinguish subcutaneous fat from visceral fat deposition. In addition, there are insufficient age- and sex-specific data that can define obesity.

ABSI, which is another obesity index, was recently introduced using WC, weight, and height. This index was closely associated with mortality in the United States. However, it has limitations in terms of its applicability to different genders and countries. Moreover, it may have statistical limitations with regard to skewness and symmetry. LBSIZ has recently been proposed to complement and improve ABSI. Our previous study has demonstrated that LBSIZ is a standard normalized obesity measurement that is independent of weight, height, and BMI, and it can be age and sex independent. Importantly, while LBSIZ was not correlated with BMI in Korean population, it had a linear relationship with CVD risks across different obesity phenotype, grouped by BMI and criteria for metabolic syndrome in the present study. This result evidently showed the usefulness of LBSIZ as a complementary mea-
sure to overcome the drawbacks of BMI.

Our findings can provide a convincing explanation for the heterogeneous results of the previous studies with regard to the risk of developing CVDs in MHO individuals.\textsuperscript{8,37,39} Taking these into consideration, the findings show that the limitation of BMI should be considered along with other determining factor, such as total body fat mass, fat distribution or body shape, when predicting cardiovascular risk.

The main strength of the present study is that it was a large-scale observational study with a representative sample. However, there were several limitations in this study as well. First, this is a cross-sectional study. To clarify the relationship between each obesity phenotype and CVD events, further prospective studies must be conducted. Second, because the present study involved a population of Korean adults, the results are applicable only in Koreans. Third, we could not assess data on mortality, and fatal CVD events may have been missed.

In conclusion, we analyzed the association between the CVD outcomes and obesity phenotype by body shape. LBSIZ had a linear relationship in individuals with MHO phenotype. These findings may have implications in terms of CVD risk assessment in MHO group. In addition, the applications of these findings are important to supplement the drawback of BMI in both clinical practice and epidemiologic studies. However, further prospective studies must be conducted to redefine MHO phenotype in view of body shape to validate MHO.

**CONFLICTS OF INTEREST**

The authors declare no conflict of interest.

**REFERENCES**

1. World Health Organization. Fact sheet on obesity and overweight [Internet]. Geneva: World Health Organization; 2017 [cited 2018 May 13]. Available from: http://www.who.int/en/news-room/fact-sheets/detail/obesity-and-overweight
2. Korean Statistical Information Service. Prevalence of obesity in Korea [Internet]. Daejeon: Statistics Korea; 2018 [cited 2018 Feb 5]. Available from: http://kosis.kr/statHtml/statHtml.do?orgId=117&tblId=DT_11702_N101#
3. Solomon CG, Manson JE. Obesity and mortality: a review of the epidemiologic data. Am J Clin Nutr 1997;66(4 Suppl): 104S-1050S.
4. Krauss RM, Winston M, Fletcher BJ, Grundy SM. Obesity: impact on cardiovascular disease. Circulation 1998;98:1472-6.
5. Moon S, Oh CM, Choi MK, Park YK, Chun S, Choi M, et al. The influence of physical activity on risk of cardiovascular disease in people who are obese but metabolically healthy. PLoS One 2017;12:e0185127.
6. Wallström P, Bjartell A, Gullberg B, Olsson H, Wirfält E. A prospective Swedish study on body size, body composition, diabetes, and prostate cancer risk. Br J Cancer 2009;100:1799-805.

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**Figure 3.** Relationship between continuous LBSIZ and the odds ratio for cardiovascular events according to obesity phenotypes. (A) Total. (B) Subgroup by obesity phenotypes. Adjusted for age, sex, family income level, education level, moderate intensity physical activities (> 120 minute per week), alcohol consumption, smoking status, and energy intake. LBSIZ, z-score of the log-transformed A Body Shape Index; MUO, metabolically unhealthy obese; MUNO, metabolically unhealthy nonobese; MHO, metabolically healthy obese; MHNO, metabolically healthy nonobese.
7. Hamer M, Stamatakis E. Metabolically healthy obesity and risk of all-cause and cardiovascular disease mortality. J Clin Endocrinol Metab 2012;97:2482-8.
8. Merkedal B, Vatten LJ, Romundstad PR, Laugsand LE, Janszky I. Risk of myocardial infarction and heart failure among metabolically healthy but obese individuals: HUNT (Nord-Trøndelag Health Study), Norway. J Am Coll Cardiol 2014; 63:1071-8.
9. Voulgari C, Tentolouris N, Dilaveris P, Tousoulis D, Katsilambros N, Stefanadis C. Increased heart failure risk in normal-weight people with metabolic syndrome compared with metabolically healthy obese individuals. J Am Coll Cardiol 2011; 58:1343-50.
10. Ogorodnikova AD, Kim M, McGinn AP, Muntner P, Khan U, Wildman RP. Incident cardiovascular disease events in metabolically benign obese individuals. Obesity (Silver Spring) 2012;20:651-9.
11. Kramer CK, Zinman B, Retnakaran R. Are metabolically healthy overweight and obesity benign conditions? A systematic review and meta-analysis. Ann Intern Med 2013;159:758-69.
12. Rey-López JP, de Rezende LF, Pastor-Valero M, Tess BH. The prevalence of metabolically healthy obesity and obesity benign conditions? A systematic review and critical evaluation of the definitions used. Obes Rev 2014; 15:781-90.
13. Romero-Corral A, Somers VK, Sierra-Johnson J, Thomas RJ, Collazo-Clavell ML, Korinek J, et al. Accuracy of body mass index in diagnosing obesity in the adult general population. Int J Obes (Lond) 2008;32:959-66.
14. Schneider HJ, Friedrich N, Klotsche J, Pieper L, Nauck M, John U, et al. The predictive value of different measures of obesity for incident cardiovascular events and mortality. J Clin Endocrinol Metab 2010;95:1777-85.
15. Flegal KM, Graubard BI, Williamson DF, Gail MH. Excess deaths associated with underweight, overweight, and obesity. JAMA 2005;293:1861-7.
16. Krakauer NY, Krakauer JC. A new body shape index predicts mortality hazard independently of body mass index. PLoS One 2012;7:e39504.
17. Chung W, Park CG, Ryu OH. Association of a new measure of obesity with hypertension and health-related quality of life. PLoS One 2016;11:e0155399.
18. Korea Centers for Disease Control and Prevention. The guideline of the forth Korea National Health and Nutrition Examination Survey (KNHANES-IV). Cheongju: Korea Centers for Disease Control and Prevention; 2009.
19. Korea Centers for Disease Control and Prevention. The guideline of the fifth Korea National Health and Nutrition Examination Survey (KNHANES-V-1). Cheongju: Korea Centers for Disease Control and Prevention; 2010.
20. WHO Expert Consultation. Appropriate body-mass index for Asian populations and its implications for policy and intervention strategies. Lancet 2004;363:157-63.
21. Jung CH, Lee WJ, Song KH. Metabolically healthy obesity: a friend or foe? Korean J Intern Med 2017;32:611-21.
22. 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) final report. Circulation 2002;106:3143-421.
23. Yoon JW, Jung CH, Kim MK, Park HE, Park KS, Jang HC, et al. Influence of the definition of “metabolically healthy obesity” on the progression of coronary artery calcification. PLoS One 2017;12:e0178741.
24. Jung CH, Lee MJ, Kang YM, Jang JE, Leem J, Hwang JY, et al. The risk of incident type 2 diabetes in a Korean metabolically healthy obese population: the role of systemic inflammation. J Clin Endocrinol Metab 2015;100:934-41.
25. Heianza Y, Arase Y, Tsuji H, Fujihara K, Saito K, Hsieh SD, et al. Metabolically healthy obesity, presence or absence of fatty liver, and risk of type 2 diabetes in Japanese individuals: Toranomon Hospital Health Management Center Study 20 (TOPICS 20). J Clin Endocrinol Metab 2014;99:2952-60.
26. Hinouhou GM, Czernichow S, Dugravot A, Gailhac A, Nabi H, Brunner EJ, Kivimaki M, et al. Metabolically healthy obesity and the risk of cardiovascular disease and type 2 diabetes: the Whitehall II cohort study. Eur Heart J 2015;36:551-9.
27. Eshtiaghi R, Keihani S, Hosseinpanah F, Barzin M, Azizi F. Natural course of metabolically healthy abdominal obese adults after 10 years of follow-up: the Tehran Lipid and Glucose Study. Int J Obes (Lond) 2015;39:514-9.
28. Rahman M, Berenson AB. Accuracy of current body mass index obesity classification for white, black, and Hispanic reproductive-age women. Obstet Gynecol 2010;115:982-8.
29. Gurunathan U, Myles PS. Limitations of body mass index as an obesity measure of perioperative risk. Br J Anaesth 2016;116:319-21.
30. Rothman KJ. BMI-related errors in the measurement of obesity. Int J Obes (Lond) 2008;32 Suppl 3:S56-9.
31. Gastaldelli A, Miyazaki Y, Pettiti M, Matsuda M, Mahankali S, Santini E, et al. Metabolic effects of visceral fat accumulation in type 2 diabetes. J Clin Endocrinol Metab 2002;87:5098-103.
32. Tran TT, Yamamoto Y, Gesta S, Kahn CR. Beneficial effects of subcutaneous fat transplantation on metabolism. Cell Metab 2008;7:410-20.
33. Coutinho T, Goel K, Corrêa de Sá D, Carter RE, Hodge DO, Kragelund C, et al. Combining body mass index with measures of central obesity in the assessment of mortality in subjects with coronary disease: role of “normal weight central obesity”. J Am Coll Cardiol 2013;61:553-60.
34. Bener A, Youssafzai MT, Darwish S, Al-Hamaq AO, Nasralla EA, Abdul-Ghani M. Obesity index that better predict metabolic syndrome: body mass index, waist circumference, waist hip ratio, or waist height ratio. J Obes 2013;2013:269038.
35. Bays H. Central obesity as a clinical marker of adiposopathy; increased visceral adiposity as a surrogate marker for global fat dysfunction. Curr Opin Endocrinol Diabetes Obes 2014;21:345-51.
36. Chan JM, Rimm EB, Colditz GA, Stampfer MJ, Willett WC. Obesity, fat distribution, and weight gain as risk factors for clinical diabetes in men. Diabetes Care 1994;17:961-9.
37. Arnlöv J, Ingelsson E, Sundström J, Lind L. Impact of body mass index and the metabolic syndrome on the risk of cardiovascular disease and death in middle-aged men. Circulation 2010;121:230-6.
38. Duncan MJ, Mota J, Vale S, Santos MP, Ribeiro JC. Associations between body mass index, waist circumference and body shape index with resting blood pressure in Portuguese adolescents. Ann Hum Biol 2013;40:163-7.
39. Hinnouho GM, Czernichow S, Dugravot A, Batty GD, Kivimaki M, Singh-Manoux A. Metabolically healthy obesity and risk of mortality: does the definition of metabolic health matter? Diabetes Care 2013;36:2294-300.