Published in final edited form as:
J Cardiometab Syndr. 2009 ; 4(1): 12–19. doi:10.1111/j.1559-4572.2008.00029.x.

Waist Circumference, Body Mass Index, and Their Association With Cardiometabolic and Global Risk

Allison H. Christian, EdD, **Heidi Mochari, MPH, RD**, and **Lori J. Mosca, MD, PhD**
From Columbia University, New York, NY

Abstract

Total body fat and adipose tissue distribution are associated with cardiometabolic risk, yet there are conflicting data as to whether waist circumference (WC) or body mass index (BMI) is a better predictor of cardiovascular risk. To determine whether WC or BMI was more strongly associated with cardiometabolic risk, family members of patients with cardiac disease were studied (N=501; mean age, 48 years; 66% female; 36% nonwhite). Height, weight, WC, BMI, blood pressure, high-density lipoprotein cholesterol, triglycerides, glucose, high-sensitivity C-reactive protein, and lipoprotein-associated phospholipase A_2 were systematically measured. Global risk was calculated using the Framingham function. Increased WC and BMI were equally strong predictors of cardiometabolic and global risk. The prevalence of cardiometabolic risk factors and their correlation with WC and BMI varied by race/ethnicity. Our data support inclusion of WC and BMI in screening guidelines for diverse populations to identify individuals at increased cardiometabolic risk.

The prevalence of obesity, a global public health problem, is reaching epidemic proportions and is of great concern because it increases risk of coronary heart disease, stroke, diabetes, certain cancers, mortality, and health care expenses.[1,2] Previous research has documented that both absolute total body fat and adipose tissue distribution are associated with cardiometabolic risk.[3–6] However, there are conflicting data as to whether fat distribution and central adiposity (assessed by waist circumference [WC]) or absolute total fat (assessed by body mass index [BMI]) is more closely associated with cardiovascular risk.[7,8]

Multiple obesity indices (ie, BMI, waist-hip ratio, WC, body fat mass) are used to define cardiometabolic risk in clinical and research settings. Initially, waist-hip ratio was the most widely used index of central fat distribution due to its benefits in routine monitoring and assessment in patients. The World Health Organization recommends using BMI, the National Cholesterol Education Program defines the metabolic syndrome using WC, and the National Heart, Lung, and Blood Institute's (NHLBI) Guidelines for the Identification, Evaluation, and Treatment of Overweight and Obesity in Adults support the combined evaluation of BMI and WC in individuals who are overweight (BMI ≥25 kg/m^2).[9–11] The NHLBI guidelines also recommend that practitioners use BMI to assess overweight and obesity and to identify those with increased disease risk.[11] Nonstandardized definitions for obesity have contributed to differences in the prevalence and incidence of the metabolic syndrome reported in previous studies. Recent research has high-lighted the association that exists between WC and cardiovascular disease (CVD) risk factors such as serum lipid levels, insulin resistance, and blood pressure, suggesting that it may be a more useful clinical indicator of risk than BMI.[12]

Address for correspondence: Lori J. Mosca, MD, PhD, Director, Preventive Cardiology, NewYork-Presbyterian Hospital, 601 West 168th Street, #43, New York, NY 10032, E-mail: ljm10@columbia.edu.

However, further data are required to determine whether BMI or WC is a better predictor of cardio-metabolic risk factors, especially among ethnically diverse samples.

The purpose of this study was to evaluate the independent association of WC and BMI with major CVD risk factors including blood pressure, high-density lipoprotein (HDL) cholesterol, triglycerides, fasting glucose, high-sensitivity C-reactive protein (hs-CRP), lipoprotein-associated phospholipase A_2 ($LpPLA_2$), and global cardiac risk among a diverse group of individuals who participated in a family-based lifestyle intervention trial at NewYork-Presbyterian Hospital. It is hypothesized that both anthropometric indices will be associated with major cardiometabolic risk factors and that those associations will vary by race/ethnicity.

Methods

Design and Participants

Participants in the Family-Based Intervention Trial for Heart Health (FIT Heart) were included in this cross-sectional analysis (N=501; mean age, 48±13 years; 66% female; 36% nonwhite). The design and main results have been published elsewhere.[13] FIT Heart is a 1-year randomized controlled clinical trial among family members/cohabitants of patients hospitalized with CVD at NewYork-Presbyterian Hospital, Columbia University Medical Center campus. Family members or cohabitants were recruited into the study, and patients were only eligible if they were candidates for the primary prevention of CVD and did not have established CVD or a CVD risk equivalent. Only 1 family member was permitted to participate in the study. The purpose of FIT Heart was to test the effectiveness of a hospital-based standardized screening and educational intervention to increase adherence to national CVD prevention goals.

Risk Factor Screening

Each participant completed a standardized questionnaire including demographic data, medical history, medication use, family history of CVD, and smoking status. Physical activity level was also assessed at baseline using standardized questions that were adapted from the Behavioral Risk Factor Survey and validated. The questions collected information about the number of days per week participants engaged in physical activity, as well as the number of minutes per day. The following questions were included: “At least once a week, do you engage in any regular physical activity (brisk walking, jogging, bicycling, etc.) long enough to work up a sweat?” “If yes, how many days per week do you engage in physical activity that works up a sweat?” “For each time you engage in physical activity, how many minutes do you exercise for?” Bilingual staff members were available to assist participants, and all forms were available in English and Spanish.

Trained health care professionals performed standardized cardiometabolic risk factor screenings including height, weight, BMI, WC, blood pressure, HDL cholesterol, triglycerides, fasting glucose, hs-CRP, and $LpPLA_2$.

Blood Pressure

Systolic and diastolic blood pressure was assessed by an automated blood pressure monitor in the Columbia University General Clinical Research Center (GCRC) using standard protocol. Briefly, the patient was sitting, with the arm not restricted by clothing and legs uncrossed. The patient was asked to relax arm muscles and rest for 5 minutes before blood pressure was measured. The patient’s arm was supported at heart level, with the palm up. Trained personnel palpated the brachial artery and positioned the cuff 1 inch above the site of brachial pulsation and the center bladder of the cuff above the artery. The fully deflated cuff was wrapped snugly around the upper arm. The research-grade automated blood pressure monitor was activated. Extra-large cuffs were available and utilized on an as-needed basis. In this study, hypertension

was defined as a systolic blood pressure ≥140 mm Hg or a diastolic blood pressure ≥90 mm Hg based on national guidelines.[14] Utilization of blood pressure medication was recorded and adjusted for in analyses when appropriate.

Body Composition

Height was measured by a precision wall-mounted standardized height rod located in each examination room of the Columbia University GCRC. Body weight was taken by research-grade, portable Healthometer scales (Jarden Consumer Solutions, Boca Raton, FL) and recorded to the nearest 1 lb. BMI was calculated directly by the standard formula: weight (kg)/height $(m)^2$. In this study, overweight was defined as a BMI 25.0 to 29.9 kg/m^2, and obesity was defined as a BMI ≥30 kg/m^2.

WC was measured by trained examiners using a US government standard protocol.[11] Measurements were recorded to the nearest 0.3 cm. In this study, increased WC was defined as >88.9 cm in women and >101.6 cm in men. Due to variations in WC measurement methodology used by various health care organizations and studies, the trained health care professionals conducting this study performed a quality assurance evaluation among a small sample of individuals attending a recent CVD screening event (n=104), and interrater (r>0.97, P<.01) and intrarater (r>0.99, P<.01) reliability was high.

Lipids, Glucose, and hs-CRP

Values of plasma glucose, lipids (total cholesterol, HDL cholesterol, low-density lipoprotein (LDL) cholesterol, and triglycerides), and hs-CRP were evaluated from venous blood that was drawn from all participants at baseline after a 6- to 12-hour fast. Determination of lipids was performed on blood collected in tubes containing ethylenediamine-tetraacetic acid and were stored and analyzed in the Columbia University GCRC at −70°C for up to 2 weeks. The Centers for Disease Control lipid quality control program certifies the GCRC core laboratory. The HDL cholesterol assay was performed on the Hitachi 912 chemical analyzer (Roche Diagnostics, Indianapolis, IN) using the HDL cholesterol plus second-generation assay for direct measurement of HDL cholesterol in human serum and plasma. Triglyceride levels were also determined by enzymatic methods. LDL cholesterol was calculated using the Friedewald equation, and if triglycerides exceed 22.22 mmol/L, LDL cholesterol was assessed using a direct measurement assay. Quantitative assay was used for the measurement of hs-CRP (diaDexus, South San Francisco, CA). It is a standard solid-phase enzyme-linked immunoabsorbent assay with intra- and inter-assay coefficients of variation <3.9% and <5.1%, respectively. The diaDexus, Inc. PLAC Test (diaDexus, South San Francisco, CA), a microplate-based enzyme immunoassay, is cleared for marketing by the US Food and Drug Administration and was used for the quantitative determination of $LpPLA_2$ in human plasma.

Based on national guidelines, dyslipidemia was defined as an HDL cholesterol level <50 mg/dL in women or <40 mg/dL in men and a triglyceride level ≥150 mg/dL.[10,15] Impaired nonfasting glucose was defined as a glucose value ≥100 mg/dL, based on the American Diabetes Association expert recommendations.[16] The cutoff for elevated hs-CRP was ≥3.0 mg/L, according to national guidelines.[17] Increased $LpPLA_2$ was defined as ≥217 mg/L, the highest quartile of results.

We certify that all applicable institutional and governmental regulations concerning the ethical use of human volunteers were followed during this research. The study was approved by the institutional review board of Columbia University Medical Center.

Data Management and Statistics

All data were collected on duplicate standardized forms and double-entered into a Microsoft Access database, then exported to SAS version 9.1 (SAS Institute, Inc., Cary, NC) for purposes of statistical analysis. Variables outside specified ranges were removed from the analysis, including hs-CRP levels ≥10 mg/L (n=34). Categorical data are presented as frequencies and percentages. Continuous data are presented as means and SDs. Comparisons across racial/ethnic groups were made using the chisquare test for categorical data and analysis of variance for continuous data. Percentage 10-year risk of fatal or nonfatal coronary heart disease was determined from the calculated Framingham risk function.[10] Regression equations were calculated with each of the risk factors (ie, HDL cholesterol, blood pressure, global risk) as dependent variables and WC and/or BMI as potential predictor variables. Models were adjusted for potential confounders including age, ethnicity, education level, medication use (when applicable to the risk factor being evaluated), and smoking status. Variables and interactions with significant contributions to the model were retained in the regression equation. Utilization of blood glucose– and/or cholesterol-lowering medication was recorded and adjusted for in analyses when appropriate. Variables already included in the Framingham risk calculation such as age, treatment for blood pressure, and smoking were not included in multivariable logistic regression models. Statistical significance was set at $P<.05$.

Results

The characteristics of the 501 participants included in the analysis are listed in Table I. The mean age was 48±13 years and participants' ages ranged from 20 to 78 years; 36% were nonwhite and more than half of the sample (66%) was female. Lipids, blood glucose, hs-CRP, $LpPLA_2$, blood pressure, BMI, WC, physical activity level, and smoking status data were available on 501 participants; mean levels are presented in Table I. Overall, mean values for each CVD risk factor assessed were in a normal range except for BMI, WC, and hs-CRP, the mean values of which exceeded optimal levels.

In this study, when using the same anthropometric index as the National Health and Nutrition Examination Survey (NHANES) to define overweight and obesity (ie, BMI), 152 (31%) of patients were classified as overweight, and 324 (65%) were classified as overweight/obese. This rate of obesity is slightly higher than the national average, which may be explained by the high participation rate by women (66%) and ethnic/racial minorities in this study (36%); these are groups that obesity affects at a disproportionate rate.[1]

The prevalence of cardiometabolic risk factors and their association with WC and BMI is shown in Table II. As demonstrated, having an increased WC (>35 in women or >40 in in men) posed similar cardiometabolic risk as having an increased BMI (≥30 kg/m^2), and elevated hs-CRP was the most commonly associated cardiometabolic risk factor with increased WC (62%) and a BMI ≥30 kg/m^2 (67%).

To determine whether WC was more effective in identifying those at risk for CVD compared with BMI, we assessed the percentage of participants with abnormal WC and normal BMI and vice versa. Overall, 17% of patients who had a normal WC (>35 in women or >40 in in men) had an abnormal BMI (≥25 kg/m^2), and 24% of those meeting the BMI goal (<25 kg/m^2) had an abnormal WC.

As shown in Table III, in a univariate logistic regression model, those with an increased WC were significantly more likely to have hypertension ($P<.01$), low HDL cholesterol ($P<.0001$), increased triglycerides ($P<.0001$), high glucose ($P<.01$), and hs-CRP ≥3.0 mg/L ($P<.0001$) compared to those with a normal WC. Participants with an increased BMI were significantly more likely to have hypertension ($P<.0001$), low HDL cholesterol ($P<.0001$), increased

triglycerides ($P<.0001$), high glucose ($P<.01$), hs-CRP ≥3.0 mg/L ($P<.0001$), and a global risk ≥10% ($P=.01$) vs those with a normal BMI. When evaluating individuals who had both a WC and BMI above nationally recommended levels, we found that they were significantly more likely to be hypertensive ($P<.0001$) and have low HDL cholesterol ($P<.0001$), increased triglycerides ($P<.0001$), high glucose ($P<.001$), hs-CRP ≥3.0 mg/L ($P<.0001$), and a global risk ≥10% ($P=.01$) compared to those who met both goals. Table IV shows the univariate associations stratified by race/ethnicity.

In a multivariable model controlling for potential confounders (Table V), both WC and BMI significantly predicted cardiometabolic risk factors after adjustment for age, race/ethnicity, education, medication use (if applicable), and smoking. Having an increased WC and/or BMI remained a significant predictor of having all 5 major cardiometabolic risk factors including blood pressure ≥140/90 mm Hg, HDL cholesterol <50 (in women) or <40 (in men) mg/dL, triglycerides ≥150 mg/dL, glucose ≥100 mg/dL, and hs-CRP ≥3.0 mg/L. However, only having a BMI ≥25 kg/m^2 or both BMI ≥25 kg/m^2 and WC >35 (in women) or >40 (in men) in predicted increased $LpPLA_2$ and a global risk of ≥10%, whereas an increased WC alone was not associated with either of these variables.

Discussion

The major findings of this study among 501 ethnically diverse family members of patients hospitalized with CVD were that (1) the prevalence of major cardiometabolic risk factors among those with abnormal WC and BMI was high and varied by race/ethnicity, (2) a strong correlate of increased WC and BMI was elevated hs-CRP, and (3) WC and BMI were equally strong independent predictors of cardiometabolic and global risk, suggesting that both may be appropriate for inclusion in national screening guidelines.

Our data show that the association between WC, BMI, and major cardio-metabolic risk factors varied by race/ethnicity. While previous research has documented higher rates of obesity among racial/ethnic minority populations, these novel data prove that an association exists between obesity and certain cardiometabolic risk factors, which suggests that the use of specific anthropometric indices may be appropriate to assess cardiometabolic and global risk among specific subpopulations. [1] For instance, WC was not a significant predictor of hypertension among nonwhites, although it did identify white patients with hypertension and BMI significantly predicted hypertension among all groups. So, clinicians caring for nonwhite patients and nonwhite patients themselves may benefit from routine monitoring of WC (vs BMI) to identify a potential problem with blood pressure. This is a key finding, since hypertension disproportionately afflicts racial/ethnic minorities, and methods to better identify patients at risk are needed.

We found hs-CRP to be a strong correlate of adiposity, with 30% of those with a BMI ≥25 kg/m^2, 39% of those with a WC >35 (in women) or >40 (in men) in, and 41% of those with a BMI ≥30 kg/m^2 having an hs-CRP level ≥3.0 mg/L. Previous research has documented a positive correlation between adipocyte size and hs-CRP levels in healthy individuals, those with obesity, and first-degree relatives of type 2 diabetic patients; this suggests that obesity may result in low-grade inflammation. [18–21] Our novel data contribute to the existing body of literature by suggesting that easily measured anthropometric indices (BMI and WC) can serve as surrogate markers of abdominal fat and adipocyte size that can be utilized to identify those at increased risk for CVD based on inflammation. Previous research lends support for the consideration of hs-CRP as a potential adjunct in future global cardiac risk calculations. [22] However, the correlation between hs-CRP and 10-year Framingham coronary heart disease risk scores has been found to be partly related to the presence of obesity.[23] Our findings support a strong correlation between hs-CRP and obesity but do not address the added value of one of

these anthropometric indices over the other in risk prediction or cost-effectiveness; this should be addressed in future research.

Our finding that WC was an equally strong, independent predictor of cardiometabolic risk when compared to BMI among an ethnically diverse group of family members/cohabitants of CVD patients concurs with previous research that has demonstrated WC to have the strongest associations with health risk indicators and multiple major CVD risk factors including blood pressure, lipid, and glucose values.[7,8,12,24–33] These data lend further support to the role of WC in predicting increased cardiometabolic risk. Associations have also been found between WC and insulin and plasma circulating oxidized LDL cholesterol, but these factors were not assessed in the current study. These data suggest that the combined evaluation of these anthropometric indices, as currently recommended in NHLBI guidelines, is optimal for identifying those at risk.[11] Furthermore, these data imply that a simple screening test that can be assessed by self-examination with reasonable accuracy, waist measurement, may potentially identify those at increased risk for CVD who might benefit from further evaluation and intervention.[34–36]

Our study has limitations that should be considered. Although a large, diverse sample was obtained, participants may be select and not representative of all family members/cohabitants of patients hospitalized with CVD. Patients agreeing to participate in research are generally from higher socioeconomic groups and have better general health, suggesting that these data may represent a best-case scenario and patients choosing not to participate may have worse cardiometabolic pro-files. Further, there may have been inadvertent misclassification of risk factor status due to measurement error, although this was minimized by developing a uniform research protocol, conducting systematic training of staff, and utilizing standardized research-grade equipment. Last, only 2 surrogate markers of adiposity were assessed in this study, and therefore correlations between cardiometabolic risk factors and other anthropometric indices such as waist-hip ratio cannot be discussed. Future studies could benefit from an objective measure of abdominal fat such as dual-energy x-ray absorptiometry, computed tomography, or magnetic resonance imaging.

The rates of obesity observed in this study are fairly consistent with national averages reported in NHANES and underscore the need for future interventions to increase awareness of overweight/obesity as a risk factor for CVD and other chronic illnesses.[1]

Conclusions

Data obtained from a CVD primary prevention trial targeted to family members/cohabitants of patients hospitalized with CVD documented that a simple, inexpensive anthropometric index, such as WC or BMI, may be useful to health care providers in the clinical office setting because of its high predictive power to identify individuals with cardiometabolic risk factors who may have heightened risk of CVD. Future research and programs should evaluate the role of inflammation in cardiometabolic risk by routinely measuring adiposity using both WC and BMI. Future CVD screening and prevention guidelines should encourage assessment of WC *and* BMI, especially among diverse populations, to identify individuals at increased cardiometabolic and global risk.

Acknowledgments

This study was funded by a National Institute of Health (NIH) Research Project Award to Dr Mosca (R01 HL 075101-01A1). This work was supported in part by the National Institutes of Health (NIH)–funded General Clinical Research Center at Columbia University, an NIH Research Career Award to Dr Mosca (K24 HL076346-01A1), and an NIH research training grant to Dr Christian (T32 HL007343-27). An educational grant was provided by diaDexus, Inc. to analyze $LpPLA_2$ data. The authors would like to acknowledge **Dana Edelman, MPH**, **Brooke Fischer,**

EdD, **Karen Ochoa, MA**, and **Syncia Sabain, MA**, of the NewYork-Presbyterian Hospital Preventive Cardiology Program for their assistance with data collection.

REFERENCES

1. Hedley AA, Ogden CL, Johnson CL, et al. Prevalence of overweight and obesity among US children, adolescents, and adults, 1999–2002. JAMA 2004;291:2847–2850. [PubMed: 15199035]
2. Lear SA, Toma M, Birmingham L, et al. Modification of the relationship between simple anthropometric indices and risk factors by ethnic backgrounds. Metabolism 2003;52:1295–1301. [PubMed: 14564681]
3. Kennel WB, Cupples LA, Ramaswami R, et al. Regional obesity and risk of cardiovascular disease; the Framingham Study. J Clin Epidemiol 1991;44:183–190. [PubMed: 1995775]
4. Manson JE, Colditz GA, Stampfer MJ, et al. A prospective study of obesity and risk of coronary heart disease in women. N Engl J Med 1990;322:882–889. [PubMed: 2314422]
5. Jousilahti P, Toumilehto J, Vartiainen E, et al. Body weight, cardiovascular risk factors, and coronary mortality. 15-year follow-up of middle-aged men and women in eastern Finland. Circulation 1996;93:1372–1379. [PubMed: 8641026]
6. Nicklas BJ, Penninx BW, Cesari M, et al. Association of visceral adipose tissue with incident myocardial infarction in older men and women: the Health, Aging, and Body Composition Study. Am J Epidemiol 2004;160:741–749. [PubMed: 15466496]
7. Ho SC, Chen YM, Woo JLF, et al. Association between simple anthropometric indices and cardiovascular risk factors. Int J Obes 2001;25:1689–1697.
8. Onat A, Avci GS, Uyarel H, et al. Measures of abdominal obesity assessed for visceral adiposity and relation to coronary risk. Int J Obes 2004;28:1018–1025.
9. World Health Organization. Obesity – Preventing and Managing the Global Epidemic: Report of a WHO Consultation on Obesity. Geneva: World Health Organization; 1998.
10. National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III). 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. p. 3143-3421.
11. National Health Institutes. Clinical guidelines on the identification, evaluation, and treatment of overweight and obesity in adults: executive summary. Expert Panel on the Identification, Evaluation, and Treatment of Overweight in Adults; Am J Clin Nutr. 1998. p. 899-917.
12. Moreira-Andres MN, del Canizo-Gomez FJ, Losa MA, et al. Comparison of anthropometric parameters as predictors of serum lipids in premenopausal women. J Endocrinol Invest 2004;27:340–347. [PubMed: 15233553]
13. Mosca L, Mochari H, Liao M, et al. A novel family-based intervention trial to improve heart health: results of a randomized controlled trial. Circ Cardiovasc Quality Outcomes 2008;1:98–106.
14. Chobanian, AV.; Bakris, GL.; Black, HR., et al. Seventh report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure; Hypertension. 2003. p. 1206-1252.
15. Mosca L, Appel LJ, Benjamin EJ, et al. Evidence-based guidelines for cardiovascular disease prevention in women. Circulation 2004;109:672–693. [PubMed: 14761900]
16. American Diabetes Association. Diagnosis and classification of diabetes mellitus. Diabetes Care 2006;29:S43–S48. [PubMed: 16373932]
17. Pearson TA, Mensah GA, Alexander RW, et al. Markers of inflammation and cardiovascular disease: application to clinical and public health practice: a statement for healthcare professionals from the centers for disease control and prevention and the American Heart Association. Circulation 2003;107:499–511. [PubMed: 12551878]
18. Bahceci M, Gokalp D, Bahceci S, et al. The correlation between adiposity and adiponectin, tumor necrosis factor alpha, interleukin-6 and high sensitivity C-reactive protein levels. Is adipocyte size associated with inflammation in adults? J Endocrinol Invest 2007;30:210–214. [PubMed: 17505154]

19. Khaodhiar L, Ling PR, Blackburn GL, et al. Serum levels of interleukin-6 and C-reactive protein correlate with body mass index across the broad range of obesity. JPEN J Parenter Enteral Nutr 2004;28:410–415. [PubMed: 15568287]
20. Kriketos AD, Greenfield JR, Peake PW, et al. Inflammation, insulin resistance, and adiposity: a study of first-degree relatives of type 2 diabetic subjects. Diabetes Care 2004;27:2033–2040. [PubMed: 15277436]
21. Festa A, D'Agostino R Jr, Williams K, et al. The relation of body fat mass and distribution to markers of chronic inflammation. Int J Obes Relat Metab Disord 2001;25:1407–1415. [PubMed: 11673759]
22. Albert MA, Glynn RJ, Ridker PM. Plasma concentration of c-reactive protein and the calculated Framingham coronary heart disease risk score. Circulation 2003;108:161–165. [PubMed: 12835213]
23. Rogowski O, Shapira I, Toker S, et al. Obesity-related correlation between c-reactive protein and the calculated 10-y Framingham coronary heart disease risk score. Int J Obes 2005;29:772–777.
24. Lopatynski J, Mardarowicz G, Szczesniak G. A comparative evaluation of waist circumference, waist-to-hip ratio, waist-to-height ratio and body mass index as indicators of impaired glucose tolerance and as risk factors for type-2 diabetes mellitus. Ann Univ Mariae Curie Sklodowska 2003;58:413–419.
25. Bosy-Westphal A, Geisler C, Onur S, et al. Value of body fat mass vs anthropometric obesity indices in the assessment of metabolic risk factors. Int J Obes 2006;30:475–483.
26. Shen W, Punyanitya M, Chen J, et al. Waist circumference correlates with metabolic syndrome indicators better than percentage fat. Obesity 2006;14:727–736. [PubMed: 16741276]
27. Sung KC, Ryu SH. Insulin resistance, body mass index, waist circumference are independent risk factor for high blood pressure. Clin Exp Hypertens 2004;26:547–556. [PubMed: 15554457]
28. Weinbrenner T, Schroder H, Escurriol V, et al. Circulating oxidized LDL is associated with increased waist circumference independent of body mass index in men and women. Am J Clin Nutr 2006;83:30–35. [PubMed: 16400046]
29. Mosca L, Edelman D, Mochari H, et al. Waist circumference predicts cardiometabolic risk among women screened during national women's heart day. J Womens Health 2006;15:24–34.
30. Kato MM, Currier MB, Villaverde O, et al. The relationship between body fat distribution and cardiovascular risk factors in patients with schizophrenia: a cross-sectional pilot study. Prim Care Companion J Clin Psychiatry 2005;7:115–120. [PubMed: 16027766]
31. Smith DA, Ness EM, Herbert R, et al. Abdominal diameter index: a more powerful anthropometric measure for prevalent coronary heart disease risk in adult males. Keabetes Obes Metab 2005;7:370–380.
32. Tanko LB, Bagger YZ, Qin G, et al. Enlarged waist combined with elevated triglycerides is a strong predictor of accelerated atherogensis and related cardiovascular mortality in postmenopausal women. Circulation 2005;111:1883–1890. [PubMed: 15837940]
33. Cassidy AE, Bielak LF, Zhou Y, et al. Progression of subclinical coronary atherosclerosis. Does obesity make a difference? Circulation 2005;111:1877–1882. [PubMed: 15837939]
34. Freudenheim JL, Darrow SL. Accuracy of self-measurement of body fat distribution by waist, hip, and thigh circumferences. Nutr Cancer 1991;15:179–186. [PubMed: 1866312]
35. Spencer EA, Roddam AW, Key TJ. Accuracy of self-reported waist and hip measurements. Public Health Nutr 2004;7:723–727. [PubMed: 15369609]
36. Rimm EB, Stampfer MJ, Colditz GA, et al. Validity of self-reported waist and hip circumferences in men and women. Epidemiology 1990;1:466–473. [PubMed: 2090285]

Table I

Characteristics of Respondents (N=501)

Characteristic	**No. (%)**	
Age, y (mean±SD)	48±13	
Female	332 (66)	
Nonwhite	178 (36)	
>High school education	386 (78)	
Employed/student	370 (74)	
Married/living with someone	328 (66)	
No health insurance	70 (14)	
Nonsmoker	448 (90)	
	Range	Mean±SD
HDL cholesterol, mg/dL	20–160	59±19
Triglycerides, mg/dL	35–624	116±67
Glucose, mg/dL	40–310	99±17
hs-CRP, mg/L	0–10	1.96±2.09
$LpPLA_2$, mg/L	41–453	191±47
Systolic BP, mm Hg	92–219	127±16
Diastolic BP, mm Hg	49–121	77±11
BMI, kg/m^2	16–52	28±6
WC, in	22–54	36±6
Days/week exercise	0–7	1.76±1.93
Minutes/day exercise	0–300	27.12±33.58

Abbreviations: BMI, body mass index; BP, blood pressure; HDL, high-density lipoprotein; hs-CRP, high sensitivity C-reactive protein; $LpPLA_2$, lipoprotein-associated phospholipase A_2; WC, waist circumference.

Table II

Prevalence of Cardiometabolic Risk Factors byWCand BMI Levels

	BP ≥140/90	HDL-C <40 (women/50 (men) mg/dL	TG ≥150 mg/dL	Glucose ≥100 mg/dL	hs-CRP[a] ≥3.0 mg/L	$LpPLA_2$ ≥217 mg/L	Global Risk[b] ≥10%
WC, >35 (women)/>40 (men) in	30%	33%	31%	49%	39%	27%	11%
BMI, ≥25 kg/m^2	28%	29%	28%	45%	30%	27%	11%
BMI, ≥30 kg/m^2	30%	35%	35%	46%	41%	28%	12%
Central adiposity[c]	28%	28%	28%	45%	29%	28%	11%

Abbreviations: BMI, body mass index; BP, blood pressure; HDL-C, high-density lipoprotein cholesterol; hs-CRP, high-sensitivity C-reactive protein; $LpPLA_2$, lipoprotein-associated phospholipase A_2; TG, triglycerides; WC, waist circumference.

[a] Excluding hs-CRP ≥10.

[b] Calculated using the Framingham function.

[c] Defined as BMI ≥25 kg/m^2 andWC>35 in (women) or 40 in (men) vs BMI <25 kg/m^2 andWC≤35 in (women) or 40 in (men).

Table III

Univariate Association Between Cardiometabolic Risk Factors and Obesity Indices, Overall

Predictor Variables	BP ≥140/90 mm Hg (yes vs no)	HDL-C <40 (women)/50 (men) mg/dL (yes vs no)	TG ≥150 mg/dL (yes vs no)	Glucose ≥100 mg/dL (yes vs no)	hs-CRP[a] ≥3.0 mg/L (yes vs no)	$LpPLA_2$ ≥217 mg/L (yes vs no)	Global Risk[b] ≥10% (yes vs no)
WC, >35 (women)/40 (men) in vs ≤35 (women)/40 (men) in	1.91 (**<.01**)	2.80 (**<.0001**)	2.48 (**<.0001**)	1.77 (**<.01**)	3.80 (**<.0001**)	1.16 (.49)	1.41 (.28)
BMI, ≥25 vs <25 kg/m^2	3.24 (**<.0001**)	4.67 (**<.0001**)	4.91 (**<.0001**)	1.80 (**<.01**)	3.29 (**<.0001**)	1.45 (.10)	4.25 (.01)
Central adiposity[c] (yes vs no)	3.03 (**<.0001**)	4.38 (**<.0001**)	4.61 (**<.0001**)	2.02 (**<.001**)	3.08 (**<.0001**)	1.50 (.07)	2.90 (.01)

Abbreviations: BMI, body mass index; BP, blood pressure; HDL-C, high-density lipoprotein cholesterol; hs-CRP, high-sensitivity C-reactive protein; $LpPLA_2$, lipoprotein-associated phospholipase A_2; TG, triglycerides; WC, waist circumference. Values are odds ratios (*P* values).

[a] Excluding hs-CRP ≥10.

[b] Calculated using the Framingham function. Boldface values represent significance at $P<.05$.

[c] Defined as BMI ≥25 kg/m^2 and WC>35 in (women) or 40 in (men) vs BMI <25 kg/m^2 and WC≤35 in (women) or 40 in (men).

Table IV

Univariate Association Between Cardiometabolic Risk Factors and Obesity Indices by Race/Ethnicity

Predictor Variables	BP ≥140/ 90 mm Hg (yes vs no)		HDL-C <40 (women)/50 (men) mg/dL (yes vs no)		TG ≥150 mg/dL (yes vs no)		Glucose ≥100 mg/dL (yes vs no)		hs-CRP[a] ≥3.0 mg/L (yes vs no)		$LpPLA_2$ ≥217 mg/L (yes vs no)		Global Risk[b] ≥10% (yes vs no)	
	White	**NW**	**White**	**NW**	**White**	**NW**	**White**	**NW**	**White**	**NW**	**White**	**NW**	**White**	**NW**
WC, >35 (women)/40 (men) in vs ≤35 (women)/40 (men in	2.08 (**<.01**)	1.71 (.14)	3.06 (**<.001**)	2.16 (**.02**)	2.24 (**<.01**)	3.78 (**<.01**)	1.84 (**.01**)	1.76 (.07)	3.82 (**<.0001**)	3.26 (**<.001**)	1.50 (.11)	0.95 (.91)	1.34 (.45)	1.87 (.28)
BMI, ≥25 vs <25 kg/m^2	2.90 (**<.001**)	5.21 (**<.01**)	7.58 (**<.0001**)	2.13 (.08)	5.12 (**<.0001**)	6.32 (**.01**)	1.98 (**<.01**)	1.54 (.24)	2.96 (**<.001**)	3.15 (**<.01**)	1.47 (.43)	4.58 (**.03**)	2.54 (**.03**)	—
Central adiposity[c] yes vs no)	2.68 (**<.01**)	5.03 (<.**01**)	7.02 (**<.0001**)	2.04 (.09)	4.72 (**<.0001**)	6.11 (**.01**)	2.25 (**<.001**)	1.69 (.16)	2.74 (**<.01**)	3.03 (**.01**)	1.52 (.10)	4.43 (**.03**)	2.37 (.05)	—

Abbreviations: BMI, body mass index; BP, blood pressure; HDL-C, high-density lipoprotein cholesterol; hs-CRP, high-sensitivity C-reactive protein; $LpPLA_2$, lipoprotein-associated phospholipase A_2; NW, nonwhite; TG, triglycerides; WC, waist circumference. Values are odds ratios (*P* value).

[a] Excluding hs-CRP ≥10.

[b] Calculated using the Framingham function. Boldface values represent significance at *P*<.05.

[c] Defined as BMI ≥25 kg/m^2 and WC>35 in (women) or 40 in (men) vs BMI <25 kg/m^2 and WC≤35 in (women) or 40 in (men).

Table V

Multivariable Association Between Cardiometabolic Risk Factors and Obesity Indices

	Outcome Variables						
Predictor Variables	**BP ≥140/90 mm Hg (yes vs no)**	**HDL-C <40 (women)/50 (men) mg/dL (yes vs no)**	**TG ≥150 mg/dL (yes vs no)**	**Glucose ≥100 mg/dL (yes vs no)**	**hsCRP[a] ≥3.0 mg/L (yes vs no)**	**$LpPLA_2$ ≥217 mg/L (yes vs no)**	**Global Risk[b] ≥10% (yes vs no)**
WC, >35 (women)/40 (men) in vs ≤35 (women)/40 (men) in	1.91 (**<.01**)	3.03 (**<.0001**)	2.99 (**<.0001**)	1.95 (**<.001**)	3.33 (**<.0001**)	1.40 (.14)	1.38 (.32)
Sex, female vs male	0.54 (**.01**)	0.64 (.07)	0.58 (**.03**)	0.45 (**<.001**)	1.82 (**.02**)	0.61 (**.03**)	NA
Age, >50 vs ≤50 y	2.09 (**<.01**)	0.46 (**<.01**)	0.64 (.08)	2.39 (**<.001**)	0.65 (0.08)	1.22 (.39)	NA
Race/ethnicity, minority vs white	1.00 (.98)	1.25 (.37)	0.53 (**.02**)	1.13 (.57)	1.62 (.05)	0.35 (**<.0001**)	0.60 (.17)
Education, ≤HS vs >HS	1.49 (.14)	1.33 (.30)	1.08 (.79)	0.85 (.51)	1.12 (.69)	1.03 (.92)	1.70 (.16)
Medication use, [c]yes vs no	1.03 (.38)	0.99 (.57)	0.99 (.54)	NA	NA	NA	NA
Smoking, yes vs no	1.66 (.25)	0.53 (.07)	0.46 (**.02**)	1.13 (.71)	2.08 (.09)	0.37 (**<.01**)	NA
BMI, ≥25 vs <25 kg/m^2	3.16 (**<.0001**)	4.06 (**<.0001**)	5.02 (**<.0001**)	1.77 (**<.01**)	3.36 (**<.0001**)	1.63 (**.04**)	3.14 (**<.01**)
Sex, female vs male	0.72 (.18)	0.88 (.62)	0.84 (.49)	0.55 (**<.01**)	2.43 (**<.001**)	0.71 (.14)	NA
Age, >50 vs ≤50 y	2.19 (**<.01**)	0.47 (**<.01**)	0.67 (.11)	2.43 (**<.001**)	0.68 (.11)	1.25 (.33)	NA
Race/ethnicity, minority vs white	0.96 (.87)	1.23 (.41)	0.50 (**.01**)	1.14 (.55)	1.59 (.06)	0.34 (**<.0001**)	0.55 (.11)
Education, ≤HS vs >HS	1.45 (.17)	1.31 (.33)	1.05 (.86)	0.88 (.60)	1.13 (.65)	1.02 (.94)	1.55 (.25)
Medication use, [c]yes vs no	1.03 (.42)	0.99 (.43)	0.99 (.42)	NA	NA	NA	NA
Smoking, yes vs no	1.72 (.22)	0.57 (.10)	0.47 (**.03**)	1.16 (.65)	2.21 (.06)	0.37 (**<.01**)	NA
Central adiposity,[d] yes vs no	2.88 (**<.001**)	3.87 (**<.0001**)	4.71 (**<.0001**)	1.99 (**<.01**)	3.09 (**<.0001**)	1.69 (**.03**)	2.94 (**.01**)
Sex, female vs male	0.70 (.14)	0.87 (.56)	0.82 (.42)	0.55 (**<.01**)	2.37 (**<.01**)	0.70 (.14)	NA
Age, >50 vs ≤50 y	2.15 (**<.01**)	0.47 (**<.01**)	0.66 (.10)	2.42 (**<.0001**)	0.67 (.10)	1.24 (.35)	NA
Race/ethnicity, minority vs white	0.98 (.92)	1.25 (.38)	0.51 (**.01**)	1.13 (.58)	1.61 (.60)	0.34 (**<.0001**)	0.56 (.12)
Education, ≤HS vs >HS	1.47 (.16)	1.32 (.32)	1.06 (.81)	0.86 (.54)	1.15 (.06)	1.02 (.96)	1.56 (.24)
Medication use,[c] yes vs no	1.03 (.42)	0.99 (.42)	0.99 (.41)	NA	NA	NA	NA
Smoking, yes vs no	1.71 (.23)	0.57 (.10)	0.47 (**.03**)	1.15 (.67)	2.19 (.06)	0.36 (**<.01**)	NA

Abbreviations: BMI, body mass index; BP, blood pressure; HDL-C, high-density lipoprotein cholesterol; HS, high school; hs-CRP, high-sensitivity C-reactive protein; $LpPLA_2$, lipoproteinassociated phospholipase A_2 NA, not adjusted for because patient was not taking medication for the variable under analysis or because the variable was included in the Framingham risk calculation; TG, triglycerides; WC, waist circumference. Values are odds ratios (*P* value).

[a]Excluding hs-CRP ≥10.

[b]Calculated using the Framingham function.

[c]Controlled for when assessing the applicable risk factor (ie, BP, cholesterol, glucose).

[d] Defined as BMI ≥25 kg/m^2 and WC>35 in (women) or 40 in (men) vs BMI <25 kg/m^2 and WC≤35 in (women) or 40 in (men). BMI and WC were not included in the model because they were highly correlated with each other (P<.0001). Boldface values represent significance at P<.05.