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# Waist circumference and not body mass index explains obesity-related health risk[1–3]

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**ABSTRACT**

**Background:** The addition of waist circumference (WC) to body mass index (BMI; in kg/m$^2$) predicts a greater variance in health risk than does BMI alone; however, whether the reverse is true is not known.

**Objective:** We evaluated whether BMI adds to the predictive power of WC in assessing obesity-related comorbidity.

**Design:** Subjects were 14 924 adult participants in the third National Health and Nutrition Examination Survey, grouped into categories of BMI and WC in accordance with the National Institutes of Health cutoffs. Odds ratios for hypertension, dyslipidemia, and the metabolic syndrome were compared for overweight and class I obese BMI categories and the normal-weight category before and after adjustment for WC. BMI and WC were also included in the same regression model as continuous variables for prediction of the metabolic disorders.

**Results:** With few exceptions, overweight and obese subjects were more likely to have hypertension, dyslipidemia, and the metabolic syndrome than were normal-weight subjects. After adjustment for WC category (normal or high), the odds of comorbidity, although attenuated, remained higher in overweight and obese subjects than in normal-weight subjects. However, after adjustment for WC as a continuous variable, the likelihood of hypertension, dyslipidemia, and the metabolic syndrome was similar in all groups. When WC and BMI were used as continuous variables in the same regression model, WC alone was a significant predictor of comorbidity.

**Conclusions:** WC, and not BMI, explains obesity-related health risk. Thus, for a given WC value, overweight and obese persons and normal-weight persons have comparable health risks. However, when WC is dichotomized as normal or high, BMI remains a significant predictor of health risk. *Am J Clin Nutr* 2004;79:379–84.

**KEY WORDS** Abdominal obesity, metabolic syndrome, hypertension, dyslipidemia

## INTRODUCTION

It has long been recognized that body mass index (BMI; in kg/m$^2$) is a predictor of the morbidity and mortality that are due to numerous chronic diseases, including type 2 diabetes, cardiovascular disease (CVD), and stroke (1, 2). In addition, it has been established that abdominal obesity, assessed by waist circumference (WC), predicts obesity-related health risk (1–4), and the weighted evidence indicates that WC coupled with BMI predicts health risk better than does BMI alone (3, 5–7). In fact, recent findings indicate that WC is a stronger marker of health risk than is BMI (4). The utility of BMI and WC in predicting obesity-related health risk has been recognized by the National Heart, Lung, and Blood Institute of the National Institutes of Health (NIH; 2). The NIH guidelines indicate that the health risk increases in a graded fashion when moving from the normal-weight through obese BMI categories, and that within each BMI category men and women with high WC values are at a greater health risk than are those with normal WC values (2). Thus, it is assumed that BMI and WC have independent effects on obesity-related comorbidity.

Although it is evident that the addition of WC to BMI predicts a greater variance in health risk than does BMI alone, whether the reverse is true is unclear. That is, for a given WC value or WC category (eg, normal or high), it is not known whether higher BMI values indicate a greater health risk than do lower BMI values. However, it has been shown that WC and hip or thigh circumference have independent and opposite effects on metabolic health risk. Whereas WC is positively associated with health risk, hip and thigh circumferences are negatively associated with health risk (8–13). This implies a protective effect of a large hip or thigh circumference (or both), which could be due to a greater lean mass in the nonabdominal regions. Indeed, lean body mass is negatively associated with all-cause mortality (14). When this fact is coupled with the knowledge that WC is a strong predictor of both abdominal and nonabdominal fat (15, 16), it seems reasonable to suggest that, for a given WC value, higher BMI values may not indicate an increased health risk. Addressing this issue could have important implications for the determination of the manner in which WC and BMI are used to predict obesity-related comorbidity in both the research and the clinical settings.

The purpose of this investigation was to determine whether BMI adds to the predictive power of WC in assessing obesity-

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related health risk. This question was addressed by using metabolic and anthropometric data from the third National Health and Nutrition Examination Survey (NHANES III), which is a large cohort representative of the US population.

## SUBJECTS AND METHODS

### Study population

NHANES III was conducted by the National Center for Health Statistics and the Centers for Disease Control and Prevention to estimate the prevalence of major diseases, nutritional disorders, and potential risk factors for these diseases (17). NHANES III was a nationally representative, 2-phase, 6-y (mid-1988 through mid-1994), cross-sectional survey. The complex sampling plan used a stratified, multistage, probability cluster design. The total sample comprised 33 199 persons. Full details of the study design, recruitment, and procedures are available from the Department of Health and Human Services (17). Of the total sample, 14 924 were persons aged ≥17 y in whom measures of WC, height, weight, and metabolic variables were obtained and who fit within the BMI categories examined (see below). Written informed consent was obtained from all participants, and the protocol was approved by the National Center for Health Statistics.

### Survey methods

#### *Body mass index and waist circumference*

Body weight and height were measured to the nearest 0.1 kg and 0.1 cm, respectively, by using standardized equipment and procedures (17, 18). WC was measured to the nearest 0.1 cm at the level of the iliac crest while the subject was at minimal respiration (17).

#### *Metabolic variables*

Three blood pressure measurements were obtained at 60-s intervals with the subject in a seated position by using a standard manual mercury sphygmomanometer (17). The average of the 3 readings was used for this analysis. Blood samples were obtained after a minimum 6-h fast for the measurement of serum cholesterol, triacylglycerol, lipoproteins, and glucose as described in detail elsewhere (17, 19). Briefly, cholesterol and triacylglycerol concentrations were measured enzymatically in a series of coupled reactions that hydrolyzed cholesterol ester and triacylglycerol to cholesterol and glycerol, respectively. Plasma glucose was assayed by using a hexokinase enzymatic method (17, 20).

#### *Confounding variables*

The confounding variables, including age, race, health behaviors (ie, alcohol, smoking, physical activity), and the ratio of poverty to income, were assessed by questionnaire. Age and poverty:income were included in the analysis as continuous variables. Poverty:income, which was calculated on the basis of family income and family size (17), was used as an index of socioeconomic status. Race was coded as 0 for non-Hispanic whites, 1 for non-Hispanic blacks, 2 for Hispanics, and 3 for other races. Alcohol consumption was graded as none (0 drinks/mo), moderate (1–15 drinks/mo), or heavy (>15 drinks/mo). Subjects were considered current smokers if they smoked at the time of the interview; previous smokers if they were not current smokers but had smoked 100 cigarettes, 20 cigars, or 20 pipefuls of tobacco in their entire life; and nonsmokers if they had smoked less than those amounts. Leisure time physical activity was graded as none (<4 times/mo), low (4–10 times/mo), moderate (11–19 times/mo), or high (>19 times/mo).

### Definition of groups and terms

Subjects were divided into 2 WC groups and 3 BMI groups according to the NIH cutoffs (2). Men and women with WC values ≤102 and ≤88 cm, respectively, were considered to have a normal WC, whereas men and women with WC values >102 and >88 cm, respectively, were considered to have a high WC. On the basis of BMI, subjects were classified as normal-weight (BMI of 18.5–24.9), overweight (BMI of 25.0–29.9), or class I obese (BMI of 30.0–34.9). Because all of those who were underweight (BMI <18.5) had normal WC values and almost all (>99%) of those with class II and III obesity (BMI of ≥35.0) had high WC values, they were excluded from the data analysis.

Hypertension was defined according to the guidelines of the Joint National Committee on Detection, Evaluation, and Treatment of High Blood Pressure—ie, systolic blood pressure ≥140 mm Hg, diastolic blood pressure ≥90 mm Hg, or the use of antihypertensive medication (21). Dyslipidemia and the metabolic syndrome were defined according to the latest National Cholesterol Education Program guidelines; that is, dyslipidemia was defined as hypercholesterolemia (total cholesterol ≥240 mg/dL), high LDL cholesterol (≥160 mg/dL), low HDL cholesterol (<40 mg/dL), and high triacylglycerol (serum triacylglycerol ≥200 mg/dL), and metabolic syndrome was defined as 3 or 4 of the following: triacylglycerol concentration ≥150 mg/dL, HDL cholesterol concentration <40 mg/dL in men or <50 mg/dL in women, blood pressure ≥130/85 mm Hg, and fasting glucose concentration ≥110 mg/dL (22). The metabolic syndrome, which is also known as syndrome X and the insulin resistance syndrome, represents a clustering of plasma lipid, glucose, and blood pressure risk factors and abdominal obesity. Although the National Cholesterol Education Program guidelines include high WC as a component of the metabolic syndrome (22), for our analysis the diagnosis of the metabolic syndrome did not include a high WC.

### Statistical analysis

The INTERCOOLED STATA 7 software program (Stata Corporation, College Station, TX) was used to properly weight the sample to be representative of the population and to take into account the complex sampling strategy of the NHANES III design. Differences in age and WC were compared between normal-weight, overweight, and class I obese subjects within each WC category by using an analysis of variance. Logistic regression analysis was used to examine the associations between BMI classification and metabolic disease. Dummy variables (eg, class I obese, 0; overweight, 1; normal weight, 2) were created to compute odds ratios (ORs) for these factors. The normal-weight BMI category was used as the reference category (OR = 1.00). The logistic regression was performed in 3 steps. In the first step (eg, partially adjusted), the ORs were adjusted for the potential confounding variables including age, health behaviors, and poverty:income. In the second step (eg, fully adjusted; model 1), the ORs were adjusted for the potential confounding variables and WC, which in this case was included in the regression model as

a dichotomous variable, so that the subjects were classified as having a normal (≤102 cm in men, ≤88 cm in women) or high (>102 cm in men, >88 cm in women) WC. In the third step (eg, fully adjusted; model 2), the ORs were adjusted for the potential cofounders and WC, which was included in the regression model as a continuous variable. Logistic regression analysis was also used to examine the independent and combined effects of BMI and WC on comorbidity; BMI and WC were entered into the regression model as continuous variables. For this analysis, the ORs were computed for each unit of increase in BMI and WC. All analyses were performed separately for men and women.

## RESULTS

The descriptive and metabolic characteristics for the entire sample of men and women are shown in **Table 1**, and a breakdown of the subjects according to WC and BMI category is shown in **Table 2**. Within both the normal and high WC categories, normal-weight subjects were older (men only) and had smaller BMIs and WCs than did overweight subjects (Table 2). The overweight subjects in turn were younger and had smaller BMI and WC values than did the obese subjects within the same WC category (Table 2). Therefore, even within the same WC category (eg, normal or high), BMI and WC values were higher in the overweight and obese persons than in those with a normal BMI.

Results of the logistic regression, which show the ORs for the various obesity-related comorbidities according to BMI category, are presented in **Table 3**. After adjustment for the confounding variables alone (eg, partially adjusted ORs), the odds for all of the comorbidities were higher for the overweight and obese men and women than for the normal-weight subjects (except for high LDL cholesterol in men). After WC was included in the regression model as a dichotomous variable (eg, normal or high WC; fully adjusted OR; model 1), the odds for many of the comorbidities remained significantly higher in the overweight and obese groups (Table 3). However, after WC was included in the regression model as a continuous variable (eg, WC value in cm; fully adjusted OR; model 2), the odds of comorbidity were no longer significantly higher in either the overweight or obese men or women (except for high triacylglycerol and hypercholesterolemia in overweight men and women, respectively) than in the normal-weight men or women. An example of this effect is shown in **Figure 1**, which illustrates the odds of the metabolic syndrome with increasing BMI category before adjustment for WC, after adjustment for WC category (normal or high), and after adjustment for WC (cm) as a continuous variable.

The results of the logistic regression in which BMI alone, WC alone, or both BMI and WC were used as continuous variables to predict the obesity-related comorbidities are shown in **Table 4**. Without exception and independent of sex, both BMI alone and WC alone were strong positive predictors of comorbidity. Because the units for BMI and WC are different, the magnitude of the ORs for BMI and WC presented in Table 4 are not directly comparable. For example, in women the odds of the metabolic syndrome were 1.15 for BMI and 1.06 for WC. Thus, for every 1.0 kg/m$^2$ increase in BMI, the odds of the metabolic syndrome increased by 15%, and for every 1.0-cm increase in WC, those odds increased by 6%. When both BMI and WC were included in the regression model, WC remained a predictor of all of the comorbidities in both men and women (except for LDL cholesterol in men). However, when both BMI and WC were included in the regression model, BMI was no longer a predictor of comorbidity in either sex (except for hypertension in men).

**TABLE 1**
Descriptive characteristics and metabolic risk factors in the total sample of normal-weight, overweight, and class I obese participants from the third National Health and Nutrition Examination Survey[1]

| | Men ($n$ = 7385) | Women ($n$ = 7539) |
|---|---|---|
| Descriptive characteristics | | |
| Age (y) | 42.2 ± 17.2 | 44.0 ± 18.4 |
| BMI (kg/m$^2$) | 25.8 ± 3.6 | 25.1 ± 4.1 |
| Waist circumference (cm) | 93.3 ± 11.3 | 85.9 ± 12.0 |
| Metabolic variables | | |
| Systolic blood pressure (mm Hg) | 125 ± 16 | 121 ± 20 |
| Diastolic blood pressure (mm Hg) | 77 ± 11 | 72 ± 11 |
| Fasting glucose (mg/dL) | 97 ± 27 | 93 ± 29 |
| Total cholesterol (mg/dL) | 200 ± 41 | 205 ± 45 |
| LDL cholesterol (mg/dL) | 128 ± 36 | 124 ± 38 |
| HDL cholesterol (mg/dL) | 46 ± 13 | 56 ± 15 |
| Triacylglycerol (mg/dL) | 151 ± 130 | 125 ± 96 |

[1] $\bar{x}$ ± SD.

## DISCUSSION

The results of this study provide compelling evidence that BMI coupled with WC does not predict an increase in obesity-related health risk better than does WC alone when the 2 values are examined on a continuous scale. Thus, overweight and obese persons have a health risk that is comparable to that of normal-weight persons with the same WC value. However, when WC is dichotomized as a normal or high-risk value according to the NIH obesity guidelines, BMI remains a significant predictor of metabolic health risk. This suggests that the obesity classification system advocated by the NIH is misleading and can be improved.

The primary finding of this study, that BMI coupled with WC did not predict obesity-related health risk better than did WC alone when these 2 anthropometric measures were examined on a continuous scale, indicates that WC, and not BMI, explains obesity-related health risk. However, when WC was dichotomized into the normal and high-risk categories advocated by the NIH, BMI remained a significant predictor of health risk. This was probably explained by the fact that, even when the subjects were in the same WC category (ie, normal values), the absolute WC values were considerably greater in the obese (98 cm in men, 85 cm in women) and overweight (94 cm in men, 83 cm in women) subjects than in the normal-weight (84 cm in men, 76 cm in women) subjects.

Although our findings provide evidence that the NIH obesity classification system is useful, they also indicate that those guidelines are misleading. Specifically, the results suggest that WC is a better marker of health risk than is BMI, and consequently a greater emphasis should be placed on WC in the obesity classification system. Furthermore, our results suggest that WC is related to health risk in a graded fashion, and consequently it would be more appropriate to have >2 risk strata for WC. Lean et al (23) proposed that WC values should be classified into 3 risk strata (<94, 94–102, and >102 cm in men; <80, 80–88, and >88 cm in women). However, BMI still predicted more variance in health risk than did WC alone, even after we subdivided the

**TABLE 2**
Comparison of age, body mass index (BMI), and waist circumference (WC) in normal-weight, overweight, and class I obese men and women within the normal and high WC categories

| | Normal WC | | | High WC | | |
|---|---|---|---|---|---|---|
| | Normal-weight group | Overweight group | Class I obese group | Normal-weight group | Overweight group | Class I obese group |
| Men ($n$ = 7385) | | | | | | |
| Number (weighted %) | 43.3 | 30.3 | 1.9 | 0.3 | 11.2 | 13.0 |
| Age (y) | 42.1 ± 20.9[1,a] | 40.9 ± 15.3[b] | 33.0 ± 12.4[c] | 67.4 ± 9.2[a] | 55.4 ± 15.5[b] | 48.3 ± 15.6[c] |
| BMI (kg/m$^2$) | 22.6 ± 1.7[a] | 26.7 ± 1.3[b] | 30.9 ± 0.8[c] | 24.4 ± 0.7[a] | 28.2 ± 1.2[b] | 32.0 ± 1.3[c] |
| WC (cm) | 83.5 ± 7.5[a] | 94.2 ± 4.9[b] | 98.2 ± 3.0[c] | 103.3 ± 1.1[a] | 105.6 ± 3.5[b] | 110.3 ± 5.6[c] |
| Women ($n$ = 7539) | | | | | | |
| Number (weighted %) | 47.9 | 9.1 | 0.6 | 6.0 | 20.7 | 15.7 |
| Age (y) | 38.7 ± 16.9[a] | 39.3 ± 16.2[a] | 35.4 ± 10.8[a] | 57.2 ± 18.7[a] | 51.5 ± 78.3[b] | 47.9 ± 17.2[c] |
| BMI (kg/m$^2$) | 21.7 ± 1.7[a] | 26.4 ± 1.1[b] | 31.1 ± 0.7[c] | 23.6 ± 1.0[a] | 22.5 ± 1.4[b] | 32.0 ± 1.4[c] |
| WC (cm) | 76.2 ± 6.0[a] | 82.9 ± 3.5[b] | 85.0 ± 2.4[b] | 91.2 ± 3.1[a] | 95.6 ± 5.4[b] | 102.5 ± 7.2[c] |

[1] $\bar{x}$ ± SD. Within each sex and WC category, values with different superscript letters are significantly different, $P \leq 0.05$ (ANOVA). Without exception, there was a significant interaction of WC and BMI category with age ($P < 0.01$).

subjects into these 3 WC categories (results not shown). Nonetheless, if WC values were stratified into 5 or 6 risk strata, much as BMIs are stratified in the current NIH guidelines (2), it is possible that WC alone could be used as an indicator of health risk and that measures of BMI would not be required. This possibility has important implications, given that most members of the population cannot readily calculate their BMI (23), and this difficulty is compounded by the inaccuracy of self-reported height and weight measurements (24, 25). Approximately 20% of adults are classified in the incorrect BMI category on the basis of self-reported height and weight (25). By comparison, only 2% of men and women are classified in the incorrect 3-tiered WC category (eg, low, moderate, or high) on the basis of self-measured WC (26).

Our finding that WC is an independent predictor of health risk contrasts with the finding of Kiernan and Winkleby (27). They examined the utility of the NIH weight loss guidelines, which are based on an algorithm that employs BMI, WC, and 8 other CVD risk factors, such as LDL cholesterol and blood pressure values (2). The results of their study indicate that 98% of adults receive the same recommendations for weight loss when the NIH algorithm (based on BMI, WC, and CVD risk factors) is used and when an algorithm based on BMI and CVD risk factors alone is used. One interpretation of this finding is that WC is not a useful

**TABLE 3**
Odds ratios (and 95% CIs) for metabolic disorders comparing normal-weight, overweight, and class I obese subjects before and after adjustment for waist circumference[1]

| | Overweight subjects | | | Class I obese subjects | | |
|---|---|---|---|---|---|---|
| | Partially adjusted[2] | Fully adjusted, model 1[3] | Fully adjusted, model 2[4] | Partially adjusted[2] | Fully adjusted, model 1[3] | Fully adjusted, model 2[4] |
| Men ($n$ = 7385) | | | | | | |
| Hypertension | 1.75 (1.44, 2.11)[5] | 1.57 (1.29, 1.91)[5] | 1.11 (0.87, 1.43) | 2.99 (2.33, 3.84)[5] | 2.53 (1.21, 5.31)[5] | 1.31 (0.79, 2.21) |
| Hypercholesterolemia | 1.57 (1.25, 1.97)[5] | 1.46 (1.17, 1.83)[5] | 1.06 (0.78, 1.44) | 1.70 (1.20, 2.41)[5] | 1.99 (1.09, 3.63)[5] | 0.75 (0.41, 1.37) |
| High LDL cholesterol | 1.39 (0.95, 2.05) | 1.33 (0.93, 1.89) | 0.95 (0.63, 1.43) | 1.59 (0.87, 2.91) | 2.07 (0.34, 12.55) | 0.75 (0.26, 2.15) |
| Low HDL cholesterol | 2.28 (1.84, 2.82)[5] | 2.07 (1.60, 2.69)[5] | 1.22 (0.90, 1.66) | 3.81 (2.68, 5.41)[5] | 3.01 (1.49, 6.07)[5] | 1.10 (0.62, 1.96) |
| High triacylglycerol | 3.11 (2.40, 4.03)[5] | 2.73 (2.12, 3.51)[5] | 1.43 (1.06, 1.95)[5] | 5.16 (3.93, 6.77)[5] | 3.86 (2.08, 7.17)[5] | 1.57 (0.93, 2.67) |
| Metabolic syndrome | 2.49 (1.81, 3.44)[5] | 1.97 (1.41, 2.77)[5] | 1.12 (0.77, 1.64) | 5.08 (3.56, 7.27)[5] | 2.67 (1.60, 4.50)[5] | 1.28 (0.60, 2.72) |
| Women ($n$ = 7539) | | | | | | |
| Hypertension | 1.92 (1.56, 2.35)[5] | 1.24 (0.94, 1.64) | 1.04 (0.78, 1.40) | 3.01 (2.28, 3.98)[5] | 1.68 (1.15, 2.47)[5] | 1.02 (0.63, 1.64) |
| Hypercholesterolemia | 2.26 (1.80, 2.82)[5] | 1.77 (1.37, 2.28)[5] | 1.44 (1.07, 1.95)[5] | 1.87 (1.46, 2.38)[5] | 1.20 (0.76, 1.91) | 0.90 (0.47, 1.73) |
| High LDL cholesterol | 2.12 (1.40, 3.21)[5] | 1.61 (1.04, 2.50)[5] | 1.26 (0.80, 1.99) | 1.80 (1.34, 2.40)[5] | 1.17 (0.75, 1.82) | 0.66 (0.33, 1.35) |
| Low HDL cholesterol | 1.78 (1.35, 2.35)[5] | 1.36 (0.91, 2.02) | 1.14 (0.74, 1.76) | 2.95 (2.18, 3.99)[5] | 1.61 (1.01, 2.57)[5] | 1.20 (0.67, 2.16) |
| High triacylglycerol | 2.76 (2.13, 3.57)[5] | 1.66 (1.19, 2.31)[5] | 1.34 (0.93, 1.91) | 3.56 (2.77, 4.57)[5] | 1.42 (0.93, 2.17) | 0.59 (0.33, 1.04) |
| Metabolic syndrome | 2.51 (1.70, 3.70)[5] | 1.66 (1.09, 2.52)[5] | 1.13 (0.70, 1.83) | 4.27 (3.06, 5.93)[5] | 2.73 (1.55, 3.80)[5] | 1.09 (0.61, 1.92) |

[1] $n$ = 14 924 adult participants from the third National Health and Nutrition Examination Survey.
[2] Adjusted for age, race, physical activity, smoking, alcohol intake, and the poverty-to-income ratio.
[3] Adjusted for waist circumference category (high or low), age, race, physical activity, smoking, alcohol intake, and the poverty-to-income ratio.
[4] Adjusted for waist circumference (continuous variable), age, race, physical activity, smoking, alcohol intake, and the poverty-to-income ratio.
[5] Significantly greater than the normal-weight BMI category, $P < 0.05$ (logistic regression).

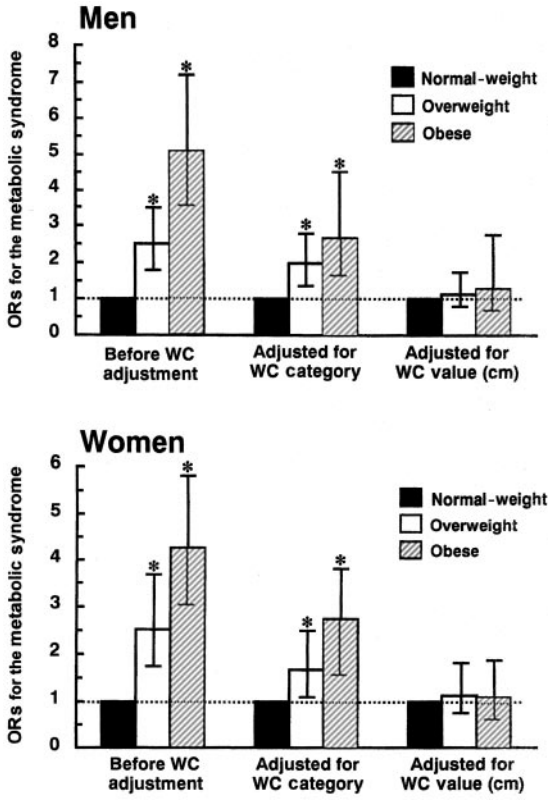


**FIGURE 1.** Odds ratios (ORs) and 95% CIs for the metabolic syndrome by BMI category before and after adjustment for waist circumference (WC) in normal-weight ($n = 3163$; reference category: OR = 1.00), overweight ($n = 3081$), and class I obese ($n = 1161$) men and normal-weight ($n = 3428$; reference category: OR = 1.00), overweight ($n = 2606$), and class I obese ($n = 1505$) women before adjustment for WC, after adjustment for WC category (normal or high), and after adjustment for WC as a continuous variable. Subjects were 14 924 adult participants from the third National Health and Nutrition Examination Survey. *Significantly greater than in the normal-weight subjects, $P < 0.01$.

clinical measure, at least within the context of the NIH weight-loss guidelines. The results of that study do not, however, indicate that WC does not add to the predictive capacity of BMI in determining the actual CVD risk factors and the risk of CVD. In fact, the results of the present study and numerous others (3, 5–7) clearly show that WC coupled with BMI predicts CVD and its risk factors better than does BMI alone. The purpose of using simple anthropometry in the identification of those at increased health risk is to identify those with CVD risk factors. Thus, it is not surprising that WC was not a significant predictor of those in need of weight management after the actual CVD risk factors were taken into account (27). In other words, the NIH weight-loss algorithm as currently presented does not permit determination of the independent contribution of WC to health risk.

Given that the subject pool of NHANES III was large and representative of the US population, that study provided perhaps the best data set with which to test our hypothesis. Even so, our study has 2 limitations that warrant recognition. First, the cross-sectional nature of this study precludes causal inferences about the associations between WC, BMI, and comorbidity. However, many studies have shown that high WC and BMI values precede the onset of morbidity and mortality (3, 7, 28, 29). The results of this study set the stage for prospective studies of these relations. Second, there was a potential bias due to survey nonresponse and the absence of values for some of the metabolic and confounding variables. However, previous NHANES studies showed little bias due to nonresponse (30).

In summary, obesity-related health risk is explained by WC and not by BMI. Thus, for a given WC value, overweight and obese persons have a health risk that is comparable with that of normal-weight persons. Future studies are required to determine whether WC alone can be used as an indicator of health risk in clinical and research settings if a greater number of WC risk strata are developed, much as are currently used for BMI. Such an expansion of WC risk strata could have important implications,

**TABLE 4**
Odds ratios (and 95% CIs) for metabolic disorders using prediction models with body mass index (BMI) alone, waist circumference (WC) alone, or both BMI and WC[1]

| | | | BMI and WC | |
|---|---|---|---|---|
| | BMI alone | WC alone | BMI | WC |
| Men ($n = 7385$) | | | | |
| Hypertension | 1.13 (1.11, 1.15)[2] | 1.04 (1.01, 1.05)[2] | 1.06 (1.00, 1.12)[2] | 1.03 (1.01, 1.05)[2] |
| Hypercholesterolemia | 1.07 (1.04, 1.10)[2] | 1.03 (1.02, 1.04)[2] | 1.02 (0.97, 1.08) | 1.02 (1.00, 1.04)[3] |
| High LDL cholesterol | 1.16 (1.12, 1.90)[2] | 1.02 (1.01, 1.14)[2] | 1.03 (0.94, 1.13) | 1.01 (0.99, 1.04) |
| Low HDL cholesterol | 1.20 (1.17, 1.23)[2] | 1.06 (1.05, 1.07)[2] | 1.03 (0.98, 1.09) | 1.05 (1.03, 1.07)[2] |
| High triacylglycerol | 1.20 (1.17, 1.23)[2] | 1.07 (1.06, 1.08)[2] | 1.05 (0.99, 1.12) | 1.05 (1.03, 1.08)[2] |
| Metabolic syndrome | 1.20 (1.15, 1.25)[2] | 1.07 (1.06, 1.08)[2] | 1.07 (0.99, 1.18) | 1.04 (1.02, 1.07)[2] |
| Women ($n = 7539$) | | | | |
| Hypertension | 1.13 (1.10, 1.15)[2] | 1.05 (1.04, 1.06)[2] | 1.02 (0.97, 1.07) | 1.05 (1.03, 1.06)[2] |
| Hypercholesterolemia | 1.08 (1.06, 1.10)[2] | 1.03 (1.03, 1.04)[2] | 1.00 (0.97, 1.04) | 1.03 (1.02, 1.05)[2] |
| High LDL cholesterol | 1.08 (1.04, 1.11)[2] | 1.04 (1.02, 1.05)[2] | 0.97 (0.92, 1.02) | 1.05 (1.02, 1.07)[2] |
| Low HDL cholesterol | 1.11 (1.08, 1.13)[2] | 1.04 (1.02, 1.05)[2] | 1.03 (0.99, 1.08) | 1.03 (1.01, 1.05)[2] |
| High triacylglycerol | 1.13 (1.11, 1.15)[2] | 1.06 (1.05, 1.07)[2] | 0.96 (0.92, 1.00) | 1.08 (1.08, 1.09)[2] |
| Metabolic syndrome | 1.15 (1.12, 1.19)[2] | 1.06 (1.05, 1.08)[2] | 1.01 (0.95, 1.07) | 1.06 (1.04, 1.09)[2] |

[1] $n = 14\,924$ adult participants from the third National Health and Nutrition Examination Survey. BMI and WC were included in the regression model as continuous variables, and the ORs were computed for each unit increase in BMI (kg/m$^2$) and WC (cm). The ORs were adjusted for age, race, physical activity, smoking, alcohol intake, and the poverty-to-income ratio.

[2] Significantly greater odds, $P < 0.05$ (logistic regression).

[3] Greater odds, $P = 0.06$ (logistic regression).

given the difficulty that most members of the public have in calculating their BMI and the inaccuracy of their findings.

IJ performed the data analysis and wrote the manuscript draft and the final article. PTK aided in the presentation and interpretation of the results and statistical analysis. RR was responsible for the study design and aided in the presentation and interpretation of the results. None of the authors declared any conflicts of interest.

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