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# Relationship between obesity and several cardiovascular disease risk factors in apparently healthy Korean individuals: comparison of body mass index and waist circumference

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**Abstract**

Recent versions of the criteria for diagnosing the metabolic syndrome have emphasized the superiority of abdominal obesity, as measured by waist circumference (WC), in identifying individuals at increased risk for cardiovascular disease (CVD). On the other hand, there is evidence that body mass index (BMI), an estimate of overall obesity, fulfills this function as effectively as does WC. The present analysis was performed to compare the relative use of these 2 indices of obesity to identify multiple CVD risk factors. The study population consisted of 19 584 apparently healthy men and women of Korean ethnicity, and the CVD risk factors measured included fasting plasma concentrations of the following variables: glucose, insulin, total, low-density lipoprotein, and high-density lipoprotein cholesterol, triglycerides, apolipoproteins A-I and B, and high-sensitivity C-reactive protein. The univariate relationships between the 2 indices of obesity and the 9 CVD risk factors were relatively modest (the highest $r$ value was 0.45), but they were all statistically significant, and the magnitude of the relationships between the CVD risk factors and BMI and WC were comparable. When multivariate analysis was performed, adjusting for age and either BMI or WC, each index of obesity continued to have an independent relationship, albeit reduced in magnitude, with the CVD risk factors. These findings suggest that measurements of BMI provide as much clinical insight as do determinations of WC in identifying multiple CVD risk factors in a large population of apparently healthy Korean men and women, and that the use of both indices would provide the most information.


## 1. Introduction

Cardiovascular disease (CVD) is known to be increased in overweight/obese individuals [1,2], and it is of clinical use to have an index of excess adiposity with which to identify those individuals at greatest CVD risk. In this context, the Adult Treatment Panel III [3] has recommended that abdominal obesity, as assessed by measuring waist circumference (WC), be used in diagnosing their version of the metabolic syndrome. The International Diabetes Federation [4] has also focused on the importance of abdominal obesity, and to satisfy their diagnostic criteria for the metabolic syndrome an individual must have a WC that exceeds an ethnic-specific value. This emphasis on the importance of WC measurements is somewhat surprising given evidence [5] from the 15 000 participants in the National Health and Nutrition Examination Survey that the correlation coefficients between measurements of body mass index (BMI) and WC were 0.9 or greater irrespective of the age, sex, and ethnicity of groups evaluated. Not only are measurements of BMI and WC highly correlated, but they were also recently shown to be correlated to a similar degree with a specific measure of insulin-mediated glucose disposal [6].

One approach to resolve these issues would be to focus on the ability of measures of obesity to actually predict adverse clinical outcomes, but even in this instance there is no definitive answer. For example, the INTERHEART study [7] presents evidence that the ratio of waist to hip girth

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(WHR) was the strongest predictor of myocardial infarction, followed in order by WC, and then BMI. In contrast, Wittchen et al [8] and Schneider et al [9] pointed out that results in the DETECT study of the development of coronary artery disease, type 2 diabetes mellitus, dyslipidemia, and hypertension in 48 353 primary care patients indicated that "waist-to-hip ratio was a weaker predictor of these disorders than WC or BMI" [8,9]. To further complicate this issue, evidence has also been presented that each of these measures of excess adiposity contributes to CVD risk [10]. Thus, although there is consensus that obesity has an adverse clinical impact, differences of opinion exist as to what index of adiposity is the most useful in identifying those individuals who are at greatest risk. The present analysis represents an attempt to provide additional insight into this problem; it was initiated to define the relationship between BMI and WC and a variety of CVD risk factors, and differs from our prior publication in several important aspects [6]. In the first place, the study is based on the findings in 19 584 apparently healthy individuals of Korean ancestry. Second, rather than examining the relationship between the 2 indices of adiposity and insulin resistance, the present analysis compared the relationship between BMI and WC and multiple metabolic risk factors for CVD. Third, in contrast to studies [11-13] that have emphasized the importance of WC in predicting adverse outcomes by "adjusting" for differences in BMI, we have also examined the impact of BMI adjusting for differences in WC.

## 2. Methods

The study population consisted of 11 599 men and 7985 women who had visited Kangbuk Samsung Hospital, College of Medicine, Sungkyunkwan University, Seoul, South Korea, between January 1, 2005, and December 31, 2005, to participate in a health screening program. By medical history, none of the participants included in this analysis had significant medical complaints, and they were without evidence of any illness on the basis of routine physical examination and laboratory evaluation. In addition, none of these individuals were taking medications to treat diabetes, hypertension, or dyslipidemia. Finally, individuals whose fasting glucose concentration was 126 mg/dL or greater were excluded from the analysis.

Height, weight, and BMI (expressed as weight in kilograms divided by square height in meters [kg/m$^2$]) were determined. Waist circumference (WC) was measured at the midlevel between the lowest rib and the iliac crest with the subject standing and breathing normally. Blood samples were collected after at least 12 hours of fasting, and plasma was separated and stored at −80°C until analyzed in the hospital clinical laboratory by the following methods. Glucose concentrations were determined by the hexokinase method (Advia 1650 Autoanalyzer, Bayer Diagnostics, Leverkusen, Germany), and insulin concentration by immunoradiometric assay (Biosource, Nivelles, Belgium), with intra- and interassay coefficients of variation of 2.1% to 4.5% and 4.7% to 12.2%, respectively. An enzymatic calorimetric test was used to measure total cholesterol (TC) and triglyceride (TG) concentrations. The selective inhibition method was used to measure the level of high-density lipoprotein cholesterol (HDL-C), and a homogeneous enzymatic calorimetric test was used to measure the level of low-density lipoprotein cholesterol (LDL-C; Advia 1650 Autoanalyzer, Bayer Diagnostics). Apolipoprotein B and apolipoprotein A-I were measured on a BN II system (Dade Behring, Marburg, Germany). High-sensitivity C-reactive protein (hs-CRP) was analyzed by performing particle-enhanced immunonephelometry, using the BN System (Dade Behring). Results were presented as milligrams per liter, and the minimum detectable hs-CRP level was 0.175 mg/L after performing 1:20 sample dilution.

Statistical analysis of the data was performed by using SPSS version 12.0 (SPSS, Chicago, IL), and continuous variable data are presented as means and standard deviations. Student *t* test was used to evaluate the differences in means, and correlation coefficients between BMI and WC with CVD risk factors were determined with Pearson correlation. CVD

Table 1
General characteristics of the study population

| | Total (N = 19 584) | Men (n = 11599) | Women (n = 7985) | *P* |
|---|---|---|---|---|
| Age (y) | 43 ± 10 | 43 ± 9 | 43 ± 10 | .220 |
| BMI (kg/m$^2$) | 24 ± 3 | 24 ± 3 | 22 ± 3 | <.001 |
| WC (cm) | 79 ± 10 | 84 ± 8 | 73 ± 8 | <.001 |
| Glucose (mg/dL) | 95 ± 9 | 97 ± 9 | 93 ± 8 | <.001 |
| Insulin ($\mu$IU/mL) | 9 ± 4 | 9 ± 4 | 9 ± 3 | .211 |
| TC (mg/dL) | 192 ± 33 | 195 ± 33 | 187 ± 34 | <.001 |
| TG (mg/dL) | 129 ± 86 | 151 ± 96 | 97 ± 57 | <.001 |
| HDL-C (mg/dL) | 53 ± 12 | 50 ± 10 | 58 ± 12 | <.001 |
| LDL-C (mg/dL) | 110 ± 28 | 114 ± 27 | 105 ± 28 | <.001 |
| TC/HDL-C | 3.75 ± 0.92 | 4.02 ± 0.89 | 3.36 ± 0.80 | <.001 |
| Apolipoprotein A-I (mg/dL) | 144 ± 24 | 140 ± 23 | 149 ± 24 | <.001 |
| Apolipoprotein B (mg/dL) | 95 ± 24 | 100 ± 22 | 88 ± 23 | <.001 |
| CRP (mg/L) | 1.18 ± 3.3 | 1.36 ± 3.7 | 0.91 ± 2.6 | <.001 |

BMI = body mass index, CRP = C-reactive protein, TC = total cholesterol, TG = triglyceride, HDL-C = high-density lipoprotein cholesterol, LDL-C = low-density lipoprotein cholesterol.

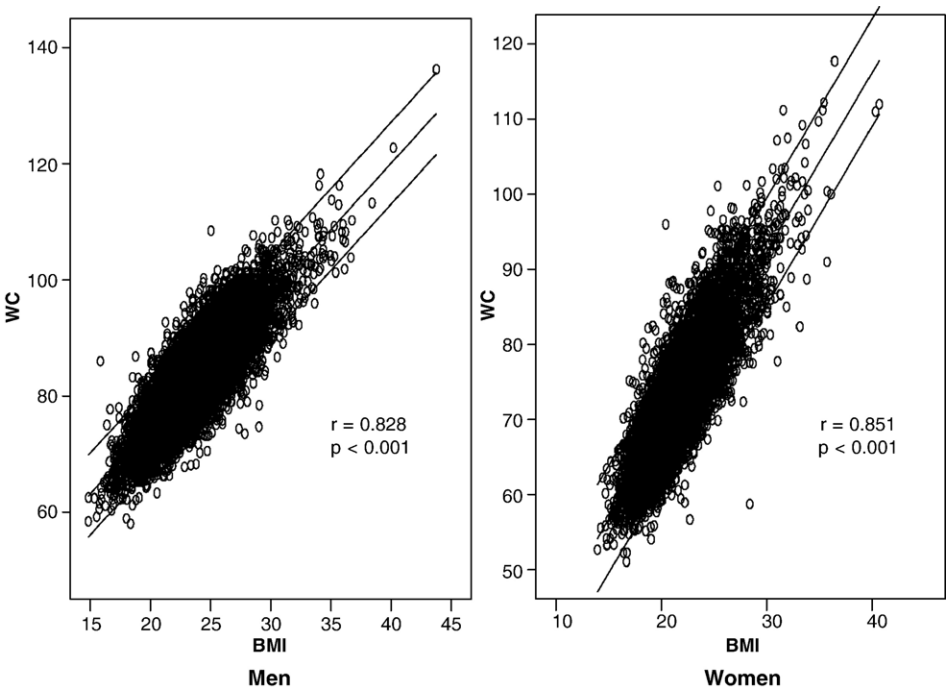


Fig. 1. Relationship between BMI and WC in men and women.

risk factors were considered as a dependent variable in an analysis of variance including BMI, WC, and the interaction BMI × WC as independent variables. All multiple comparisons were conducted with the Bonferroni correction. When the interaction BMI × WC had a $P$ value greater than .05 based on type III sums of squares, the analysis was repeated with only BMI and WC as independent variables. TG and hs-CRP concentrations were not normally distributed, so they were converted to logs and then analyzed. $P$ values less than .05 were considered statistically significant.

## 3. Results

The general characteristics of the study population are listed in Table 1. The 2 genders were not different in terms of age and plasma insulin concentrations, but men had significantly higher values for BMI, WC, glucose, TC, LDL-C, TG, apolipoprotein B, and hs-CRP concentrations ($P < .001$). In contrast, women had higher HDL-C and apolipoprotein A-I concentrations ($P < .001$).

Fig. 1 depicts the relationship between BMI and WC in the total population. There was an approximate 2-fold (WC) to 2.3-fold (BMI) variation of values for these indices in the group as a whole. The correlation coefficients ($r$) between the 2 indices of adiposity were highly correlated ($r = 0.85$) in the group as a whole, with comparable values in both men (0.83, $P < .001$) and women (0.85, $P < .001$).

Although WC and BMI measurements were highly correlated, it can be seen from Fig. 1 that the values of either measure of adiposity can account only for approximately 70% of the variability in the other estimate. To address this issue directly, we divided both men and women into quintiles of either BMI or WC, and then defined within that quintile the standard deviation of the other measure of

Table 2
Variability of the relationship between the 2 indices of obesity

A

| BMI (kg/m$^2$) | n | Mean WC (cm) ± SD |
|---|---|---|
| Men (N = 11 599) | | |
| 1 (15.3-22.2) | 2323 | 75 ± 5.0 |
| 2 (22.3-23.7) | 2323 | 80 ± 4.4 |
| 3 (23.8-25.0) | 2282 | 84 ± 4.3 |
| 4 (25.1-26.6) | 2355 | 87 ± 4.4 |
| 5 (26.7-44.2) | 2316 | 92 ± 5.8 |
| Women (N = 7985) | | |
| 1 (14.4-19.7) | 1605 | 64 ± 4.1 |
| 2 (19.8-21.1) | 1581 | 68 ± 4.1 |
| 3 (21.2-22.5) | 1607 | 72 ± 4.5 |
| 4 (22.6-24.4) | 1597 | 76 ± 4.7 |
| 5 (24.5-41.1) | 1595 | 83 ± 6.8 |

B

| WC (cm) | n | Mean BMI (kg/m$^2$) ± SD |
|---|---|---|
| Men (N = 11 599) | | |
| 1 (58-77) | 2367 | 21.3 ± 1.8 |
| 2 (77-82) | 2287 | 23.3 ± 1.5 |
| 3 (82-85) | 2307 | 24.4 ± 1.5 |
| 4 (85-90) | 2353 | 25.5 ± 1.6 |
| 5 (90-136) | 2285 | 27.6 ± 2.3 |
| Women (N = 7985) | | |
| 1 (51-65) | 1612 | 19.1 ± 1.4 |
| 2 (65-69) | 1591 | 20.6 ± 1.4 |
| 3 (70-74) | 1593 | 21.8 ± 1.6 |
| 4 (74-79) | 1603 | 23.2 ± 1.7 |
| 5 (79-118) | 1586 | 26.0 ± 2.6 |

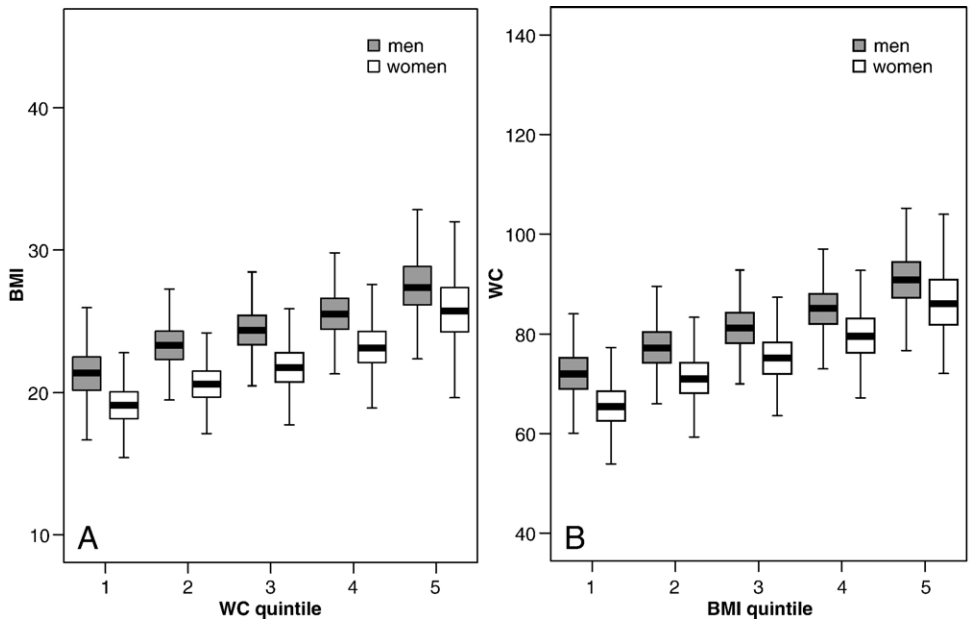


Fig. 2. Boxplots showing (A) variation of BMI values in each WC quintile and (B) variation of WC values in each BMI quintile.

adiposity. The results of this analysis are shown in Table 2. Thus, it can be seen in Table 2A that there was considerable variation in the standard deviation of WC values within each BMI quintile, and in Table 2B that there was a comparable degree of variability of BMI standard deviation values within each WC quintile.

The variability of the relationship between the 2 indices of obesity is also clearly seen in Fig. 2. It is apparent that the WC values increase with each quintile of BMI, as do BMI values with increasing quintiles of WC. In both instances, the values for men are higher than for women, but the variability of the relationship between the 2 indices of adiposity does not seem to differ as a function of gender.

Table 3 compares the relationships between the 2 indices of adiposity and multiple-risk CVD risk factors in men and women At the simplest level, the results indicate that there was a highly statistically significant relationship ($P < .001$) between both indices of adiposity and every CVD risk factor measured, Second, it can be seen that the correlation coefficients ($r$) between the 2 indices of adiposity and the CVD risk factors were quite comparable. However, it should be emphasized that the statistical significance of correlation coefficients also depends on the number of individuals being studied, and relationships of considerable statistical significance may not be of great biological importance. For example, the strongest relationships between differences in index of obesity and specific CVD risk factor were with insulin, TG, and apolipoprotein B concentrations, and the plasma concentration ratio of TC/HDL-C. However, in none of these instances could differences in BMI or WC account for more than 10% to 20% in the variability of these CVD risk factors.

The data in Table 4 indicate that the magnitude of the correlation coefficients ($r$) between either index of obesity and CVD risk factor decreased by 50% or more when

Table 3
Relationships ($r$ values) between the 2 indices of obesity (BMI and WC) and CVD risk factors

| | Male | | Female | |
|---|---|---|---|---|
| | BMI | WC | BMI | WC |
| Glucose | 0.20 | 0.23 | 0.29 | 0.29 |
| Insulin | 0.45 | 0.42 | 0.32 | 0.30 |
| TC | 0.20 | 0.21 | 0.27 | 0.29 |
| Log TG | 0.35 | 0.36 | 0.37 | 0.40 |
| HDL-C | −0.24 | −0.23 | −0.22 | −0.25 |
| LDL-C | 0.19 | 0.19 | 0.31 | 0.32 |
| TC/HDL-C | 0.35 | 0.34 | 0.40 | 0.44 |
| Apolipoprotein A-I | −0.12 | −0.11 | −0.07 | −0.04 |
| Apolipoprotein B | 0.30 | 0.31 | 0.39 | 0.43 |
| Log CRP | 0.25 | 0.27 | 0.33 | 0.33 |

All $P < .001$.

Table 4
Relationships ($r$ values) between the 2 indices of obesity (BMI and WC) and CVD risk factors after adjusting for age and the other index of obesity

| | Male | | Female | |
|---|---|---|---|---|
| | BMI | WC | BMI | WC |
| Glucose | 0.06 | 0.07 | 0.08 | 0.05 |
| Insulin | 0.16 | 0.13 | 0.14 | 0.09 |
| TC | 0.07 | 0.06 | 0.05 | 0.04* |
| Log TG | 0.09 | 0.13 | 0.06 | 0.12 |
| HDL-C | −0.08 | −0.06 | −0.02** | −0.12 |
| LDL-C | 0.09 | 0.05 | 0.08 | 0.05 |
| TC/ HDL-C | 0.13 | 0.09 | 0.06 | 0.15 |
| Apolipoprotein A-I | −0.04 | −0.04 | −0.06 | 0.01*** |
| Apolipoprotein B | 0.11 | 0.08 | 0.07 | 0.10 |
| Log CRP | 0.07 | 0.09 | 0.10 | 0.09 |

BMI, adjusting for age and WC; WC, adjusting for age and BMI. All $P$ values were less than .001, with 3 exceptions as indicated by the asterisks.

* $P = .01$.

** $P = .009$.

*** $P = .38$.

Table 5
Mean and confidence intervals of CVD risk factors according to BMI and WC status (age-adjusted mean)

| | Group 1 | Group 2 | Group 3 | Group 4 | Multiple comparison | *P* for interaction |
|---|---|---|---|---|---|---|
| | Normal BMI, normal WC | Normal BMI, high WC | High BMI, normal WC | High BMI, high WC | | |
| Men | n = 6728 | n = 235 | n = 2607 | n = 2029 | | |
| Glucose (mg/dL) | 95 (95-95) | 97 (96-98) | 98 (97-98) | 99 (99-99) | 1≠2, 1≠3, 1≠4, 2≠4, 3≠4 | .796 |
| Insulin (μIU/mL) | 7.8 (7.7-7.9) | 9.6 (9.1-10.0) | 9.6 (9.5-9.7) | 11.6 (11.5-11.8) | 1≠2, 1≠3, 1≠4, 2≠4, 3≠4 | .422 |
| TC (mg/dL) | 191 (190-192) | 201 (197-206) | 201 (199-201) | 203 (202-204) | 1≠2, 1≠3, 1≠4 | <.001 |
| TG (mg/dL) | 131 (129-133) | 170 (158-182) | 167 (164-171) | 190 (186-194) | 1≠2, 1≠3, 1≠4, 2≠4, 3≠4 | <.001 |
| HDL-C (mg/dL) | 51.7 (51.5-52.0) | 48.8 (47.5-50.1) | 48.6 (48.2-48.9) | 47.0 (46.5-47.4) | 1≠2, 1≠3, 1≠4, 3≠4 | .051 |
| LDL-C (mg/dL) | 110 (110-111) | 118 (115-122) | 118 (117-119) | 120 (118-121) | 1≠2, 1≠3, 1≠4 | .002 |
| TC/HDL-C | 3.80 (3.77-3.83) | 4.24 (4.13-4.35) | 4.23 (4.20-4.27) | 4.41 (4.37-4.45) | 1≠2, 1≠3, 1≠4, 2≠4, 3≠4 | <.001 |
| Apolipoprotein A-I (mg/dL) | 142 (141-142) | 138 (135-141) | 139 (138-140) | 136 (135-137) | 1≠2, 1≠3, 1≠4, 3≠4 | .387 |
| Apolipoprotein B (mg/dL) | 96 (95-96) | 105 (102-107) | 105 (106-106) | 109 (108-110) | 1≠2, 1≠3, 1≠4, 2≠4, 3≠4 | <.001 |
| CRP (mg/L) | 1.23 (1.14-1.42) | 1.51 (1.04-1.98) | 1.41 (1.27-1.55) | 1.71 (1.66-1.87) | 1≠4, 3≠4 | .301 |
| Women | n = 6219 | n = 495 | n = 337 | n = 934 | | |
| Glucose (mg/dL) | 92 (92-93) | 95 (94-95) | 94.8 (94-96) | 96 (96-97) | 1≠2, 1≠3, 1≠4, 2≠4, 3≠4 | .191 |
| Insulin (μIU/mL) | 8.3 (8.2-8.4) | 9.62 (9.3-9.9) | 10.2 (9.9-10.5) | 11.5 (11.2-11.7) | 1≠2, 1≠3, 1≠4, 2≠4, 3≠4 | .830 |
| TC (mg/dL) | 185 (184-186) | 192 (189-194) | 193 (190-196.) | 195 (193-197) | 1≠2, 1≠3, 1≠4 | .046 |
| TG (mg/dL) | 90 (89-92) | 111 (106-115) | 116 (110-121) | 125 (122-129) | 1≠2, 1≠3, 1≠4, 2≠4, 3≠4 | <.001 |
| HDL-C (mg/dL) | 58.8 (58.5-59.1) | 54.1 (53.0-55.2) | 55.4 (54.1-56.6) | 53.1 (52.2-53.9) | 1≠2, 1≠3, 1≠4, 3≠4 | .012 |
| LDL-C (mg/dL) | 103 (102-104) | 110 (108-112) | 111 (108-114) | 114 (112-115) | 1≠2, 1≠3, 1≠4 | .032 |
| TC/HDL-C | 3.25 (3.24-3.27) | 3.66 (3.59-3.72) | 3.60 (3.52-3.67) | 3.80 (3.75-3.85) | 1≠2, 1≠3, 1≠4, 2≠4, 3≠4 | <.001 |
| Apolipoprotein A-I (mg/dL) | 150 (149-151) | 147 (144-149) | 146 (144-149) | 145 (144-147) | 1≠2, 1≠4 | .258 |
| Apolipoprotein B (mg/dL) | 85 (85-86) | 94 (92-96) | 93 (91-96) | 97 (96-99) | 1≠2, 1≠3, 1≠4, 2≠4, 3≠4 | .004 |
| CRP (mg/L) | 0.77 (0.70-0.83) | 1.07 (0.83-1.30) | 1.08 (0.81-1.36) | 1.73 (1.56-1.90) | 1≠4, 2≠4, 3≠4 | .812 |

≠ indicates two groups are different. Multiple comparison shows no significant difference between the normal-BMI, high-WC group and high-BMI, normal-WC group. Normal BMI, <25 kg/m$^2$. Normal WC, <90 cm in men, <80 cm in women.

adjusted for age and the other index of obesity, but every correlation coefficient remained statistically significant in men. Thus, it can be concluded that in men both BMI and WC are independent predictors of the CVD risk factors measured. The findings in women were reasonably similar, but in this instance, WC was no longer an independent predictor of apolipoprotein A-I.

Table 5 presents the results of our effort to evaluate the individual impact of each index of adiposity on the CVD risk factors measured, as well as the degree of interaction between them. The data show that 78% (6219/7985) of women were nonobese by both indices compared to 58% (6728/11599) in men. Furthermore, there were relatively fewer women who had both a high BMI and a high WC (12%) than was the case in men (17%). It was of interest that relatively few individuals with a normal BMI had a high WC, with values of 2% and 6% in men and women, respectively.

Table 5 also contains multiple comparisons between the 4 adiposity categories, and it seems excessive to point out every significant difference between these groups. However, certain generalizations seem worthy of emphasis. In the first place, each CVD risk factor was significantly less in those with a normal WC and BMI compared with the other 3 groups. Second, in no instance was there a significant difference in CVD risk factor between those who were abnormal in only index (only an abnormal BMI or only an abnormal WC). Third, in most instances, CVD risk related to concentrations of glucose, insulin, TG, TC/HDL-C, and apolipoprotein B were significantly elevated in those with both a high BMI and a high WC, compared with those with only one index of obesity considered to be high. Finally, in 6 instances there was significant interaction between BMI and WC in both genders and modulation of CVD risk factors, whereas there was no interaction noted in 4 instances. Of note, the 4 variables in which significant interaction could not be discerned between the 2 indices of adiposity included concentrations of glucose, insulin, apolipoprotein A-I, and CRP.

## 4. Discussion

Perhaps the most physiologically relevant finding of the present study was that the relationships between the 2 indices of adiposity and the CVD risk factors measured were quite comparable. Furthermore, the relationship of either index of adiposity with the 9 CVD risk factors was modest in magnitude. The most robust univariate relationship (Table 3) in men was between fasting insulin concentration and BMI ($r$ = 0.45) and WC ($r$ = 0.42), whereas in women it was between the TC/HDL-C ratio and BMI ($r$ = 0.40) and WC ($r$ = 0.44). In most other instances, the differences in adiposity accounted for less than 10% of the variability in the values of the CVD risk factors measured. In light of the large number of individuals studied, it was not too surprising that there were statistically significant univariate relationships between either index of obesity and CVD risk factor, but it is apparent that the associations were not particularly strong.

On the other hand, the impact of obesity on CVD risk factors increases when both indices of excess adiposity obesity are present in the same individual. Thus, the results in Table 5 clearly demonstrate that essentially every CVD risk factor was significantly accentuated in those individuals that had abnormal values for both BMI and WC. Furthermore, in the case of TC, LDL-C, HDL-C, TC/HDL-C, TG, and apolipoprotein B concentrations, there were statistically significant interactions between the 2 indices of adiposity.

Turning now to the results that directly address the goal of the study, it is difficult to conclude from these findings that differences in abdominal obesity (WC) are more predictive of CVD risk factors than differences in overall obesity (BMI). In the first place, the 2 variables were closely related in both men and women, similar to the relationships between BMI and WC in the National Health and Nutrition Examination Survey population [5]. Second, the data in Table 3 indicate that the relationship between a given CVD risk factor and the 2 indices of adiposity were quite comparable, and although substantially lower when adjusted for age and the other obesity index, in essentially all instances each index remained significantly associated with the CVD risk factors measured. Furthermore, the relationship between WC and CVD risk factor when adjusted for differences in BMI was no different than when the impact of BMI was adjusted for differences in WC. Finally the results in Table 5 demonstrate that individuals in which only one of the indices of adiposity is abnormal have values for all the CVD risk factors that are not different from those subjects in whom only the other index is abnormal.

The results of this analysis indicate that the values of either BMI or WC provide essentially identical information as to the presence of the 9 CVD risk factors measured. Measurements of WHR were not performed, so we cannot compare the ability of this index of obesity to predict CVD risk factors. However, as pointed out in the Introduction, there are conflicting conclusions as to the relative importance of the various indices of obesity to predict adverse clinical outcomes [8-10]. In this context, there is a widely held perception that abdominal obesity, particularly the amount of visceral fat, is primarily responsible for the adverse effects of obesity [11-16]. If this were the case, WC and WHR should be superior predictors of the untoward effects of obesity as compared to BMI because they provide a more specific estimate of abdominal obesity. However, it is clear from the data presented that WC was no more useful as a predictor of CVD risk than was BMI. Furthermore, if abdominal obesity, per se, is the most important contributor to the adverse impact of obesity, it is not obvious why WHR was a more powerful predictor of myocardial infarction than was WC in the INTERHEART study [8]. Indeed, the observation that WHR is a better predictor of CVD events than WC seems to be largely because the greater the hip girth, the *less* likely was myocardial infarct to occur. These findings are clearly not supportive of the notion that increases in visceral obesity are primarily responsible for the adverse consequences of obesity. It should also be noted that there is evidence that differences in both BMI and WC are significantly associated to variations in multiple fat depots, including visceral fat [17].

In conclusion, the results of the present study in apparently healthy Korean adults have shown that having an ethnic-specific value for either an abnormal BMI or WC identifies individuals at increased CVD risk. In addition, the relationship between the individual indices of obesity and each CVD risk factor was comparable. Furthermore, the nature of these relationships was similar to those observed in studies of other Asian populations [18,19]. On the other hand, CVD risk was accentuated when an abnormal BMI and WC were both present. The observation that individuals at greatest CVD risk are identified when both measures of obesity are abnormal is consistent with a number of recent publications [18-21], based on findings in several different ethnic groups, and in children, adolescents, and adults. The clinical importance of making both measurements is highlighted by the finding in Table 5 that only 2% of the men in this study had an isolated abnormal WC, whereas 40% had either an abnormal BMI by itself, or combined with an abnormal WC. Perhaps the simplest conclusion from these and related studies is to emphasize that measurements of either overall (BMI) and abdominal (WC) obesity can help identify those individuals at increased risk to develop a number of related clinical syndromes. Indeed, rather than to continue controversies as to which of the various indices is *superior*, it seems that the most clinically relevant information is gained when subjects have abnormal values for *both* BMI and WC.

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