Alcohol consumption and the metabolic syndrome in Korean adults: the 1998 Korean National Health and Nutrition Examination Survey1,2

Yeong Sook Yoon, Sang Woo Oh, Hyun Wook Baik, Hye Soon Park, and Wha Young Kim

ABSTRACT

Background: The metabolic syndrome is associated with a high risk of cardiovascular disease morbidity and mortality. Light and moderate alcohol consumption have been associated with reduced cardiovascular disease morbidity and mortality.

Objective: This study was performed to examine the association between alcohol consumption and the metabolic syndrome.

Design: The study sample comprised 7962 Korean adults (3597 men, 4365 women) who had participated in the 1998 Korean National Health and Nutrition Examination Survey.

Results: The prevalence of the metabolic syndrome was 20.8% in men and 26.9% in women. The adjusted odds ratio for the metabolic syndrome in the group consuming 1–14.9 g alcohol/d was 0.71 (95% CI: 0.53, 0.95) in men and 0.80 (95% CI: 0.65, 0.98) in women. Alcohol consumption had a significant inverse relation with the odds ratio for low HDL cholesterol in all alcohol groups. Heavy alcohol consumption (≥30 g/d) was associated with significantly higher odds ratios for high blood pressure and high triacylglycerol in men and high fasting blood glucose and high triacylglycerol in women. Odds ratios for the metabolic syndrome and its components tended to increase with increasing alcohol consumption. The dose-response relation of the odds ratio between alcohol consumption and the clustering of ≥3 risk factors was significant in both the high and low HDL-cholesterol groups.

Conclusions: Although alcohol consumption had a significant inverse relation with the odds ratio for low HDL cholesterol in all alcohol groups, an increasing dose-response relation was found between alcohol consumption and the odds ratio for the metabolic syndrome. This might be due to the opposite relation of alcohol consumption to other components of the metabolic syndrome. Am J Clin Nutr 2004;80:217–24.

KEY WORDS Alcohol, metabolic syndrome, HDL cholesterol, blood pressure, triacylglycerol, glucose, waist circumference

INTRODUCTION

The metabolic syndrome is characterized by abdominal obesity, hypertension, dyslipidemia, and elevated fasting blood glucose and is associated with insulin resistance and compensatory hyperinsulinemia. Importantly, the metabolic syndrome predicts a high risk for the future development of type 2 diabetes and coronary artery disease (1–3). A recent cohort study found an increased risk of both cardiovascular disease morbidity and all-cause mortality in patients with the metabolic syndrome (4).

Although the mechanism underlying the development of the metabolic syndrome is not understood fully, it has been proposed that the metabolic syndrome appears as a result of the reciprocal action of several environmental factors, such as diet, smoking, alcohol consumption, and physical activity, in individuals with a genetic predisposition that is not yet well known. An important reason environmental factors should be considered is that they are potentially modifiable. In particular, alcohol consumption is one of the most prevalent habits in the general population of Koreans. Light to moderate alcohol consumption is associated with a reduction in the risk of cardiovascular disease mortality (5), which has been increasing recently in Korea.

The dose-response relation between alcohol consumption and risk of coronary artery disease is J- or U-shaped, suggesting that the risk of coronary artery disease is greatest when alcohol consumption is high, lowest when alcohol consumption is low or moderate, and increased in persons who do not consume alcohol at all (6, 7). The results of several epidemiologic studies (6, 8, 9) on the relation between the risk of coronary artery disease, death, and alcohol consumption have shown that light to moderate alcohol consumption has a protective role and might reduce the risk of coronary artery disease and stroke. A meta-analysis of the relation between alcohol consumption and risk of coronary artery disease concluded that there was a 20% reduction in risk when alcohol consumption was between 0 and 20 g alcohol/d (7).

The beneficial effect of regular, light to moderate alcohol consumption on the development of coronary artery disease can be explained by several factors, including increases in HDL cholesterol (10) and the balance between blood coagulation and fibrinolysis (11). The harmful effects of heavy alcohol consumption are due to an increase in plasma triacylglycerol (12) and...
increased blood pressure (13, 14). Each of these factors is a component of the metabolic syndrome. Therefore, it is of interest to evaluate the overall associations of alcohol consumption with the development of the metabolic syndrome. Because the aforementioned studies of alcohol consumption were almost exclusively limited to white subjects, and similar studies of Asians are rare, we evaluated the association between alcohol consumption and the metabolic syndrome in Korean adults.

SUBJECTS AND METHODS

Subjects

This study was based on the 1998 Korean National Health and Nutrition Examination Survey (KNHNES). The KNHNES is divided into 4 parts: the Health Interview Survey, the Health Behavior Survey, the Nutrition Survey, and the Health Examination Study.

In the 1998 KNHNES, a stratified, multistage probability sampling design was used. The sampling frame was based on the 1995 National Census Registry. There were 219,771 primary sampling units, each of which contained 60 households. Two hundred sampling frames (13,523 households) from primary sampling units were randomly sampled, and 39,060 individuals from these sampling frames were included in the Health Interview Survey. The response rate of the Health Interview Survey was 90.8%. One of 3 Health Interview Survey samples was selected and the Health Examination, Health Behavior, and Nutrition Surveys were conducted on selected samples. The survey was completed by 9,771 of 10,876 individuals who participated in the Health Examination Study, and data from 7,962 participants aged ≥20 y were used in this analysis.

The KNHNES is a national survey jointly conducted by the Korea Institute for Health and Social Affairs and the Korea Health Industry Development Institute and commissioned by the Ministry of Health and Welfare in response to the regulations in the National Health Promotion Act. The data used in this study were originally produced by the Korea Institute for Health and Social Affairs and the Korea Health Industry Development Institute and were reanalyzed with their permission.

The Health Interview and Health Behavior Surveys

The Health Interview and Health Behavior Surveys included well-established questions to determine the demographic and socioeconomic characteristics of the subjects. These included questions on age, education level, occupation, income, marital status, smoking habits, alcohol consumption, exercise, previous and current diseases, and family disease history.

Smoking status was divided into 3 categories: current smoker, ex-smoker, and nonsmoker. The total number of packs of cigarettes smoked was calculated from the total number of years spent smoking multiplied by the number of cigarettes smoked daily, divided by 20.

Subjects were questioned about whether they exercised with an intensity that left them with slight difficulty in breathing and sweating. Subjects who exercised regularly at a moderate intensity were asked about the frequency at which they exercised per week and the length of time per exercise session.

Alcohol consumption was assessed by questioning the subjects about their drinking behavior during the month before the interview. The subjects were asked about their average frequency (days per month) and amount (in mL) of alcoholic beverages ingested on a typical occasion or during a typical day.

The average amount and number of alcoholic beverages consumed was converted into the amount of pure alcohol (in g) consumed per day. For the analysis, the subjects were categorized into 4 groups according to average daily alcohol consumption: nonconsumers, light consumers (1.0–14.9 g alcohol/d), moderate consumers (15.0–29.9 g alcohol/d), and heavy consumers (≥30 g alcohol/d) (8, 15). Men in the heavy alcohol group were further divided into 2 subgroups: 30–79.9 and ≥80 g alcohol/d. We did not separate the female heavy consumers into 2 groups because only 9 women consumed ≥80 g alcohol/d.

Nutrition Survey

Daily energy and nutrient intakes were assessed by using a 24-h recall method and a food intake frequency method from the Nutrition Survey.

Health Examination Study

Height, body weight, and waist circumference were measured during the Health Examination Study. Height was measured to the nearest 0.1 cm on a Seriter stadiometer (850–2060 mm; Holtain Ltd, Crymych, United Kingdom) with the subject standing barefoot. Body weight was measured to the nearest 0.1 kg on a balanced scale (Holtain Ltd, Crymych, United Kingdom) with an adjustable height bar. Body mass index (Quetelet’s BMI) was calculated as follows: BMI = weight (kg)/height squared (m^2). Waist circumference was measured to the nearest 0.1 cm at the narrowest point between the lowest rib and the uppermost lateral border of the right iliac crest.

Blood pressure was measured with a mercury sphygmomanometer (Baumanometer; WA Baum Co Inc, New York) after the subject had rested for 5 min in a sitting position. Study subjects refrained from smoking or ingesting caffeine for 30 min before the measurement. The first appearance of sound (phase 1 Korotkoff sound) was used to define systolic blood pressure and the disappearance of sound (phase 5 Korotkoff sound) was used to define diastolic blood pressure (16). Two readings each of systolic and diastolic blood pressure were recorded, and the average of each measurement was used for data analysis. If the first 2 measurements differed by >5 mm Hg, additional readings were obtained.

Blood samples were collected from the antecubital vein to measure serum concentrations of total cholesterol, triacylglycerol, HDL cholesterol, and glucose after 10–12 h of starvation. All biochemical analyses were carried out within 2 h of blood sampling. Total cholesterol, triacylglycerol, HDL cholesterol, and glucose were measured by enzymatic methods with a Hitachi 747 autoanalyzer (Hitachi Instruments Inc, Tokyo) and commercially available kits [AUTO T-18 cholesterol kit, AUTO TAG kit, and CHOLETEST HDL kit (EMBIEL Co Ltd, Gunpo, Korea) and SICDIA GLZYME kit (Shinyang Chemical Co Ltd, Pusan, Korea)].

Definition of the metabolic syndrome

We used the 2001 definition of the metabolic syndrome suggested by the National Cholesterol Education Program Adult Treatment Panel III (17). The metabolic syndrome was defined as 3 or more of the following 5 risk factors: 1) abdominal obesity
(waist circumference > 90 cm for men and > 80 cm for women),
2) serum triacylglycerol ≥150 mg/dL, 3) serum HDL cholesterol
<40 mg/dL for men and <50 mg/dL for women, 4) systolic/
diastolic blood pressure ≥130/85 mm Hg, and 5) fasting plasma
glucose ≥110 mg/dL.

Because Asians have a greater risk of fitting the metabolic
profile at lower waist circumferences than do whites (17), the
assessment of abdominal obesity on the basis of waist circum-
ference as defined by the National Cholesterol Education Pro-
gram Adult Treatment Panel III (>102 cm for males, >94 cm for
females) does not apply to Asians. Therefore, we used the ab-
dominal obesity guidelines for waist circumference suggested by
the 1998 World Health Organization Asian Pacific Guideline
(18) for the cutoff at which waist circumference increased the
risk of obesity-related disease. Subjects treated for diabetes mel-
litus or hypertension were included in the study.

Statistics

We used SAS (version 8.0; SAS Institute Inc, Cary, NC) for all
statistical analyses, and P values <0.05 were considered to be
statistically significant.

Demographic variables, alcohol intake, anthropometric vari-
ables, and laboratory data differed between the men and the
women. The interaction of sex and amounts of alcohol consumed
day with the metabolic syndrome and its components and the
clustering of risk factors in each HDL-cholesterol group were
significant (P for interaction <0.05) except for high triacylglyc-
erol (P for interaction = 0.06). Thus, we analyzed the data sepa-
rately by sex.

Differences among groups in age, marital status, education
level, smoking status, and exercise relative to the amount of
alcohol consumed were analyzed with a chi-square test. Analysis
of variance with Tukey’s post hoc test was used to determine the
significance of differences in age, household income, number of
packs of cigarettes smoked per year, waist circumference, BMI,
frequencies of alcohol consumption, energy intake, percentage
of energy from fat, and each of the components of the metabolic
syndrome according to the amount of alcohol consumed per day.

Odds ratios were calculated by using multiple logistic regres-
sion analysis to evaluate the associations between the metabolic
syndrome and individual components of the metabolic syndrome
and amounts of alcohol consumed per day after adjustment for
age, BMI, education level, income, marital status, smoking sta-
tus, exercise, and percentage of energy from fat. Multiple logistic
regression analysis was used to analyze the associations between
clustering of risk factors and amounts of alcohol consumed per
day after adjustment for age, BMI, education level, income,
marital status, smoking status, exercise, and percentage of en-
ergy from fat in each HDL-cholesterol group. Tests of linear
trend were performed by scoring the categories of alcohol intake
and entering the score as a continuous term in the regression
model.

RESULTS

Characteristics of the study subjects

Data from a total of 7962 subjects (3597 men and 4365
women) were analyzed. The average ages of the men and the
women were 44.2 ± 14.8 and 45.1 ± 16.0 y, respectively. Within
the study group, 81.6% of the men and 52.4% of the women were
currently consuming alcohol, and the mean amounts of alcohol
consumed per day were 30.1 ± 38.4 g for men and 6.6 ± 13.2 g
for women. The metabolic syndrome was prevalent in 20.8% of
the men and 26.9% of the women. The proportions of study
subjects treated for hypertension and type 2 diabetes, respec-
tively, were 6.1% and 3.8% for men and 8.7% and 3.6% for
women. The characteristics of the subjects according to alcohol
consumption are shown in Table 1.

Metabolic and anthropometric variables of subjects
according to alcohol consumption

In a comparison of heavy (≥30 g alcohol/d) and light (1–14.9
alcohol/d) alcohol consumption, men who consumed alcohol
heavily (30.0–79.9 and ≥80 g/d) had significantly higher values
for mean waist circumference, systolic blood pressure, diastolic
blood pressure, triacylglycerol, HDL cholesterol, and fasting
blood glucose (Table 2). Women who consumed alcohol heavily
(≥30 g/d) had significantly higher systolic and diastolic blood
pressure, triacylglycerol, and HDL cholesterol. Compared with
nonconsumers, male light alcohol consumers had significantly
lower systolic blood pressure and fasting blood glucose, and
female light alcohol consumers had lower values for systolic
blood pressure, diastolic blood pressure, triacylglycerol, and
fasting blood glucose.

The metabolic syndrome and its components according to
alcohol consumption

The odds ratios for the metabolic syndrome and its compo-
nents for subjects who consumed alcohol are shown in Table 3.
The odds ratio for the metabolic syndrome was significantly
lower among subjects who consumed 1–14.9 g alcohol/d (men:
OR = 0.71, 95% CI: 0.53, 0.95; women: OR = 0.80, 95% CI:
0.66, 0.98). In the men, the odds ratios for high blood pressure
were significantly elevated in the groups consuming 30–79.9 g
alcohol/d (OR = 1.45, 95% CI: 1.12, 1.87) and ≥80 g alcohol/d
(OR = 1.88; 95% CI: 1.32, 2.68), whereas in the women, the odds
ratio for high blood pressure was elevated in the groups consum-
ing 15–29.9 g alcohol/d (OR = 1.71; 95% CI: 1.10, 2.64). The
odds ratio of high triacylglycerol was significantly reduced in
female light alcohol consumers and significantly elevated in both
male (30.0–79.9 and ≥80 g/d) and female (≥30 g/d) heavy
alcohol consumers. The odds ratios for low HDL cholesterol
was significantly reduced in both men and women in all alcohol
categories. The odds ratio of high fasting blood glucose was
significantly elevated in women consuming ≥30 g alcohol/d
(OR = 2.12; 95% CI: 1.13, 3.97). There were significant dose-
response relations between alcohol consumption and the metab-
olic syndrome and its components. Except for high fasting
blood glucose in men, the odds ratios for the metabolic syndrome
and its components increased with increasing alcohol
consumption.

The odds ratios for the metabolic syndrome and its compo-
nents according to the frequency of alcohol consumed compared
with the odds ratios in nonconsumers are not shown because
there were high correlations between the amount of alcohol con-
sumed and the frequency of alcohol consumption (men: r = 0.68,
P < 0.0001; women: r = 0.72, P < 0.0001). In addition, the
results based on the frequency of alcohol consumption were
similar to those based on the amounts consumed.
<table>
<thead>
<tr>
<th>Characteristics of the subjects according to alcohol consumption</th>
<th>Men</th>
<th>Women</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (y)</td>
<td>52.1 ± 15.8a,b</td>
<td>50871</td>
</tr>
<tr>
<td>20–29 [n (%)]</td>
<td>70 (11)</td>
<td>1155</td>
</tr>
<tr>
<td>30–39 [n (%)]</td>
<td>86 (13.5)</td>
<td>1155</td>
</tr>
<tr>
<td>40–49 [n (%)]</td>
<td>122 (19.2)</td>
<td>1155</td>
</tr>
<tr>
<td>50–59 [n (%)]</td>
<td>127 (20)</td>
<td>1155</td>
</tr>
<tr>
<td>≥ 60 [n (%)]</td>
<td>230 (36.2)</td>
<td>1155</td>
</tr>
<tr>
<td>Income (× 10⁷ Won/mo)</td>
<td>118.6 ± 94.7b</td>
<td>145.6 ± 100.9b</td>
</tr>
<tr>
<td>Education [n (%)]</td>
<td>62 (9.8)</td>
<td>1154</td>
</tr>
<tr>
<td>Smoker (pack year)</td>
<td>26.9 ± 24.3b</td>
<td>14.9 b</td>
</tr>
<tr>
<td>Energy intake (kcal/d)</td>
<td>2093.2 ± 778.7b</td>
<td>1802.6 ± 768.2</td>
</tr>
<tr>
<td>Fat intake (% of energy)</td>
<td>14 ± 7.4a</td>
<td>16.2 ± 8.5b</td>
</tr>
</tbody>
</table>

a,b,c: Means in the same row with different superscript letters are significantly different, P < 0.05 (ANOVA with Tukey’s post hoc test).

1 Won, Korean currency. Means in the same row with different superscript letters are significantly different, P < 0.05 (ANOVA with Tukey’s post hoc test).

2 Obtained by ANOVA for continuous variables and by chi-square test for categorical variables.

3 x ± SD (all such values).
TABLE 2
Metabolic and anthropometric variables according to alcohol consumption

<table>
<thead>
<tr>
<th></th>
<th>None</th>
<th>1–14.9 g/d</th>
<th>15–29.9 g/d</th>
<th>30–79.9 g/d</th>
<th>≥ 80 g/d</th>
<th>P²</th>
</tr>
</thead>
<tbody>
<tr>
<td>Height (cm)</td>
<td>167.5 ± 6.5a</td>
<td>169.2 ± 6.2b</td>
<td>168.7 ± 6.2c</td>
<td>168.5 ± 6.3a</td>
<td>167.9 ± 6.6a</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>64.1 ± 10.5a</td>
<td>66.3 ± 9.7b</td>
<td>66.2 ± 9.4b</td>
<td>66.4 ± 9.8a</td>
<td>65.7 ± 11.1b</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>22.8 ± 3.1a</td>
<td>23.1 ± 2.9ab</td>
<td>23.2 ± 2.8ab</td>
<td>23.4 ± 3.0b</td>
<td>23.2 ± 3.2b</td>
<td>&lt;0.006</td>
</tr>
<tr>
<td>WC (cm)</td>
<td>82.4 ± 8.7a</td>
<td>82.2 ± 8.8a</td>
<td>83 ± 7.7b</td>
<td>83.9 ± 8.1b</td>
<td>84.1 ± 9.4b</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>SBP (mm Hg)</td>
<td>128.8 ± 19.4a</td>
<td>125.6 ± 16.6b</td>
<td>128.9 ± 18.6c</td>
<td>128.8 ± 18.1b</td>
<td>134.7 ± 21.1c</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>DBP (mm Hg)</td>
<td>79.9 ± 10.9a</td>
<td>79.8 ± 10.8a</td>
<td>81.6 ± 11.6b</td>
<td>82.3 ± 12.1b</td>
<td>84.4 ± 14.8b</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>TG (mg/dL)</td>
<td>127.5 ± 61.8a</td>
<td>128.4 ± 59.5b</td>
<td>140.5 ± 71.8b</td>
<td>147.2 ± 68.9b</td>
<td>158.6 ± 75.8b</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>HDL-C (mg/dL)</td>
<td>44.7 ± 11.2a</td>
<td>46.6 ± 11.7b</td>
<td>49.2 ± 12.3b</td>
<td>50.9 ± 12.5b</td>
<td>53.4 ± 14.3b</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>FBG (mg/dL)</td>
<td>105.3 ± 39.8a</td>
<td>103.1 ± 30.2b</td>
<td>102.5 ± 31.9b</td>
<td>104.0 ± 30.3b</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

All values are ± SD. WC, waist circumference; SBP, systolic blood pressure; DBP, diastolic blood pressure; TG, triacylglycerol; HDL-C, HDL cholesterol; FBG, fasting blood glucose. Means in the same row with different superscript letters are significantly different, P < 0.05 (ANOVA with Tukey's post hoc test).

1 Obtained by ANOVA.

TABLE 3
Odds ratios (ORs) for metabolic syndrome (MS) and its components according to alcohol consumption

<table>
<thead>
<tr>
<th></th>
<th>None</th>
<th>1–14.9 g/d</th>
<th>15–29.9 g/d</th>
<th>30–79.9 g/d</th>
<th>≥ 80 g/d</th>
<th>P for trend</th>
</tr>
</thead>
<tbody>
<tr>
<td>MS²</td>
<td>1</td>
<td>0.71 (0.53, 0.95)</td>
<td>0.88 (0.63, 1.24)</td>
<td>0.87 (0.63, 1.19)</td>
<td>1.07 (0.71, 1.63)</td>
<td>&lt;0.005</td>
</tr>
<tr>
<td>High blood pressure</td>
<td>1</td>
<td>0.96 (0.76, 1.21)</td>
<td>1.29 (0.98, 1.69)</td>
<td>1.45 (1.12, 1.87)</td>
<td>1.88 (1.32, 2.68)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>High TG</td>
<td>1</td>
<td>0.89 (0.71, 1.14)</td>
<td>1.09 (0.83, 1.44)</td>
<td>1.35 (1.07, 1.75)</td>
<td>1.76 (1.23, 2.50)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Low HDL</td>
<td>1</td>
<td>0.73 (0.57, 0.92)</td>
<td>0.46 (0.34, 0.63)</td>
<td>0.29 (0.22, 0.40)</td>
<td>0.26 (0.17, 0.41)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>High FBG</td>
<td>1</td>
<td>0.86 (0.66, 1.12)</td>
<td>1.19 (0.88, 1.60)</td>
<td>0.92 (0.69, 1.23)</td>
<td>0.94 (0.64, 1.39)</td>
<td>NS</td>
</tr>
<tr>
<td>Large waist</td>
<td>1</td>
<td>0.85 (0.58, 1.27)</td>
<td>0.79 (0.52, 1.20)</td>
<td>1.08 (0.74, 1.59)</td>
<td>2.02 (1.22, 3.34)</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

All values are ± SD. WC, waist circumference; SBP, systolic blood pressure; DBP, diastolic blood pressure; TG, triacylglycerol; HDL-C, HDL cholesterol; FBG, fasting blood glucose. MS, metabolic syndrome; MS², metabolic syndrome trend. Odds ratios for MS and its components were calculated using multivariate logistic regression analysis. MS was defined as the presence of ≥ 3 of the following: high blood pressure (systolic blood pressure ≥ 130 mm Hg or diastolic blood pressure ≥ 85 mm Hg), high TG (TG ≥ 150 mg/dL), low HDL [HDL-C < 40 mg/dL (men) or <50 mg/dL (women)], high FBG (glucose ≥ 110 mg/dL), and large waist (waist circumference ≥ 90 cm (men) or ≥ 80 cm (women)). ORs for MS were determined by multiple logistic regression analysis after adjustment for age (10-y intervals), BMI (in kg/m²: ≤18.5, 18.6–22.9, 23–24.9, 25–29.9, or ≥ 30), smoking status (nonsmoker, ex-smoker, or current smoker), moderate exercise (none, irregular, or regular), household income (continuous variable), marital status (unmarried, married, widowed, or divorced), and fat intake/total energy intake.

95% CIs in parentheses. MS, metabolic syndrome; TG, triacylglycerol; HDL-C, HDL cholesterol; FBG, fasting blood glucose. Interactions between sex and amount of alcohol consumed per day were significant.

5 Defined as ≥ 3 of the following: high blood pressure (systolic blood pressure ≥ 130 mm Hg or diastolic blood pressure ≥ 85 mm Hg), high TG (TG ≥ 150 mg/dL), low HDL [HDL-C < 40 mg/dL (men) or <50 mg/dL (women)], high FBG (glucose ≥ 110 mg/dL), and large waist (waist circumference > 90 cm (men) or > 80 cm (women)). ORs for MS were determined by multiple logistic regression analysis after adjustment for age (10-y intervals), BMI (in kg/m²: ≤18.5, 18.6–22.9, 23–24.9, 25–29.9, or ≥ 30), smoking status (nonsmoker, ex-smoker, or current smoker), moderate exercise (none, irregular, or regular), household income (continuous variable), marital status (unmarried, married, widowed, or divorced), and fat intake/total energy intake.
adjusment for age (10-y interval), BMI (in kg/m
15 g alcohol/d) in Korean adults. In addition, a dose-
synctrome is negatively associated with light alcohol consump-

DISCUSSION

for clustering of Table 4. The result showed significantly increased odds ratios for clustering of ≥3 risk factors in the group consuming ≥80 g alcohol/d in men regardless of HDL-cholesterol group. There was a significant dose-response relation between the amount of alcohol consumed and clustering of risk factors in both the high and low HDL-cholesterol groups.

Table 4: Odds ratios (ORs) for clustering of risk factors according to alcohol consumption and HDL-cholesterol group.

<table>
<thead>
<tr>
<th>No. of risk factors</th>
<th>None</th>
<th>1–14.9 g/d</th>
<th>15–29.9 g/d</th>
<th>30–79.9 g/d</th>
<th>≥80 (men) or ≥30 (women) g/d</th>
<th>P for trend</th>
</tr>
</thead>
<tbody>
<tr>
<td>Men</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Low HDL group</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>≥2</td>
<td>1</td>
<td>0.75 (0.49, 1.15)</td>
<td>1.75 (1.01, 3.05)</td>
<td>1.79 (1.01, 3.17)</td>
<td>1.62 (0.65, 4.03)</td>
<td>NS</td>
</tr>
<tr>
<td>≥3</td>
<td>1</td>
<td>1.24 (0.71, 2.16)</td>
<td>2.98 (1.55, 5.73)</td>
<td>1.52 (0.74, 3.11)</td>
<td>4.36 (1.68, 11.31)</td>
<td>&lt;0.005</td>
</tr>
<tr>
<td>High HDL group</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>≥2</td>
<td>1</td>
<td>0.96 (0.70, 1.32)</td>
<td>1.05 (0.73, 1.51)</td>
<td>1.47 (1.06, 2.06)</td>
<td>1.92 (1.25, 2.94)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>≥3</td>
<td>1</td>
<td>0.93 (0.56, 1.54)</td>
<td>1.15 (0.66, 2.00)</td>
<td>1.60 (0.98, 2.61)</td>
<td>2.31 (1.28, 4.16)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Women</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Low HDL group</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>≥2</td>
<td>1</td>
<td>0.82 (0.63, 1.06)</td>
<td>1.00 (0.49, 2.06)</td>
<td>—</td>
<td>2.94 (0.94, 9.17)</td>
<td>&lt;0.005</td>
</tr>
<tr>
<td>≥3</td>
<td>1</td>
<td>0.76 (0.55, 1.03)</td>
<td>1.06 (0.45, 2.48)</td>
<td>—</td>
<td>3.80 (0.99, 14.49)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>High HDL group</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>≥2</td>
<td>1</td>
<td>0.92 (0.68, 1.24)</td>
<td>1.68 (0.91, 3.09)</td>
<td>—</td>
<td>2.33 (0.96, 5.67)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>≥3</td>
<td>1</td>
<td>1.04 (0.67, 1.61)</td>
<td>2.10 (0.96, 4.61)</td>
<td>—</td>
<td>3.65 (1.25, 10.67)</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

Interactions between sex and amount of alcohol consumed per day were significant. ORs were determined by multiple logistic regression analysis after adjustment for age (10-y interval), BMI (in kg/m²: <18.5, 18.5–22.9, 23–24.9, 25–29.9, or ≥30), smoking status (nonsmoker, ex-smoker, or current smoker), moderate exercise (none, irregular, or regular), household income (continuous variable), marital status (unmarried, married, widowed, or divorced), and fat intake/total energy intake.

Risk factors were as follows: high blood pressure (systolic blood pressure ≥130 mm Hg or diastolic blood pressure ≥85 mm Hg), high triacylglycerol (triaclyglycerol ≥150 mg/dL), high fasting blood glucose (glucose ≥110 mg/dL), and large waist [waist circumference >90 cm (men) or >80 cm (women)].

HDL <40 mg/dL (men) or <50 mg/dL (women).
HDL ≥40 mg/dL (men) or ≥50 mg/dL (women).

Odds ratios of clustering of risk factors by HDL-cholesterol group

The odds ratios for clustering of risk factors according to alcohol consumption by HDL-cholesterol group are shown in Table 4. The result showed significantly increased odds ratios for clustering of ≥3 risk factors in the group consuming ≥80 g alcohol/d in men regardless of HDL-cholesterol group. There was a significant dose-response relation between the amount of alcohol consumed and clustering of risk factors in both the high and low HDL-cholesterol groups.

DISCUSSION

The results of the present study suggest that the metabolic syndrome is negatively associated with light alcohol consumption (1–15 g alcohol/d) in Korean adults. In addition, a dose-response relation was found for the odds ratios for the metabolic syndrome and increasing alcohol consumption.

Controversial results have been reported on the relation between alcohol consumption and the prevalence of the metabolic syndrome (19–21). Dixon et al (19) showed that light to moderate alcohol consumption (defined as 0–100 gwk) has a favorable effect on fasting triacylglycerol, glucose, hemoglobin A1C, and insulin resistance in severely obese patients (BMI ≥35). However, in the Atherosclerosis and Insulin Resistance study of 391 healthy 58-y-old men, no significant difference was found in alcohol consumption between the subjects with the metabolic syndrome and those without risk factors (20).

We found that the subjects who consumed the most alcohol had higher serum HDL-cholesterol concentrations; this was true in both men and women. This result agrees well with the results of previous studies comprising different subject groups (10, 22–28). Although the relation of alcohol consumption with the metabolic syndrome, which includes HDL cholesterol, was expected to be similar to the relation of alcohol consumption with HDL cholesterol, this was prominent only in light alcohol consumers. To clarify the reason for this discrepancy, we analyzed the associations of alcohol consumption with each component of the metabolic syndrome.

The results of the present study agree with several other studies showing a J-shaped relation of alcohol intake to blood pressure, although some studies have shown only a threshold increased pressure at heavier drinking (29, 30). The seventh report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure (30) recommended that daily alcohol consumption be limited to ≤30 g for hypertensive men and ≤15 g for women and lighter-weight persons. This recommendation was based on the fact that such amounts of alcohol do not elevate blood pressure and have been associated with a lower risk of coronary artery disease. Our study also showed that blood pressure is higher in heavy alcohol consumers (those consuming ≥30 g alcohol/d) than in light consumers (Table 2). Light consumers also had lower systolic blood pressure than did nonconsumers among both men and women. This inverse relation was independent of past drinking.

Alcohol can elevate plasma triacylglycerol acutely (31), but reports of the effects of long-term, moderate alcohol consumption are inconsistent (6). When consumed in amounts >60 g/d, alcohol has a hypertriacylglycerolemic effect, and triacylglycerol concentrations increase by 0.19 mg/dL per gram of alcohol consumed per day (6), a finding that agrees with our results. Other studies showed an inverse relation between long-term, moderate alcohol consumption and fasting triacylglycerol (21, 32, 33). In the present study, the odds ratio of a high triacylglycerol
concentration was lower in light alcohol consumers (1–15 g alcohol/d) than in nonconsumers, although this difference was not significant in men.

The results of studies relating alcohol consumption to glucose and the risk of type 2 diabetes are conflicting (34–37) because of differences in the volume of alcohol consumed and the assessment methods used (35–37). One study of middle-aged and elderly men showed that the relative risk of type 2 diabetes was 0.6 (95% CI: 0.44, 0.91) in subjects consuming 30–49 g alcohol/d compared with nonconsumers (37). Holbrook et al (35) showed that men with greater alcohol consumption had an increased risk of type 2 diabetes, whereas women did not. However, our findings showed an increased odds ratio for high fasting blood glucose in women consuming ≥30 g alcohol/d compared with nonconsumers; however, this was not the case in men. There are some difficulties in directly comparing our study results with others, because the definitions for high blood glucose used in each study were different.

The results of several epidemiologic studies relating alcohol consumption to obesity do not agree (38–40). Sakuri et al (41) found that alcohol consumption had a positive relation with waist-to-hip ratio but no significant relation with body mass index, whereas Liu et al (38) suggested that alcohol consumption did not increase the risk of obesity. We found that abdominal obesity was positively associated with alcohol consumption.

As shown in Table 3, although the odds ratios for the metabolic syndrome and non-HDL components of the metabolic syndrome (except for high fasting blood glucose in men) tended to increase with increased alcohol consumption, the odds ratio for the metabolic syndrome in heavy alcohol consumers was not significantly high. This might be the result of a masking effect of HDL cholesterol, which is a crucial component of the metabolic syndrome. We thus analyzed our results by HDL-cholesterol group to clarify this. These results showed a significant dose-response relation between amount of alcohol consumed and clustering of risk factors in both the high and the low HDL-cholesterol groups. We also analyzed the odds ratios of clustering of nonlipid components of the metabolic syndrome according to the amount of alcohol consumed per day. These results showed significantly increased odds ratios for clustering of nonlipid components of the metabolic syndrome in men consuming ≥80 g alcohol/d (OR = 2.59; 95% CI: 1.26, 5.33) and in women consuming ≥30 g alcohol/d (OR = 3.79; 95% CI: 1.42, 10.09) (data not shown).

One consideration in studies of alcohol consumption is that ex-drinkers who stopped drinking because of health problems were included in the nonconsumers group. Ex-drinkers constituted 258 (40.6%) of the 635 male and 156 (7.7%) of the 2024 female nonconsumers. When all dependent variables were compared between the participants who never drank and the ex-drinkers, the ex-drinkers were found to be older and to have a lower household income than the never-drinkers (P <0.05) in men and to have lower diastolic blood pressure in women. Participants who never drank were more likely to be ex-smokers than were ex-drinkers (P <0.001 for both men and women). Persons who had quit drinking alcohol because of “worsening health” numbered 176 (68.2%) men and 88 (56.4%) women. Therefore, the lower odds ratios associated with light to moderate alcohol consumption might be exaggerated when less healthy individuals are included as nondrinkers. However, even when reanalyzed after the exclusion of ex-drinkers, the odds ratio of the metabolic syndrome was not significantly different from the one shown in Table 3 and was significantly lower among subjects who consumed 1–14.9 g alcohol/d (men: OR = 0.60, 95% CI: 0.43, 0.85; women: OR = 0.79, 95% CI: 0.65, 0.97).

Our study has some limitations. First, the design was cross-sectional. Second, recall bias might have been introduced because some data were collected by interview. However, there are also distinct advantages to our data. First, our study subjects were randomly sampled and are representative of the Korean population. Second, we assessed multiple variables associated with alcohol consumption and the metabolic syndrome by using a logistic regression model and direct interviews. Third, we measured the metabolic risk factors and anthropometric variables directly.

In summary, the results of the present study suggest that the metabolic syndrome is negatively associated with light alcohol consumption but that a dose-response relation exists between alcohol consumption and the odds ratio for the metabolic syndrome, although alcohol consumption had a significant inverse relation with low HDL-cholesterol in all alcohol groups. This was due to the opposite relation to other components of the metabolic syndrome. Light alcohol consumption might have favorable effects, but whether to recommend the consumption of alcohol should be decided only after careful consideration of the health risks, because heavy alcohol consumption is related to the aggravation of other metabolic profiles such as triacylglycerol, blood pressure, and blood glucose. In view of public health, further study of the associations between the metabolic syndrome and coronary artery disease and the mechanism of alcohol’s effects are required. In conclusion, we should not recommend current nonconsumers to drink alcohol and need not discourage Korean adults who are light consumers from consuming alcohol.

We thank Sung Ho Beck for proofreading the manuscript. We also thank the Korea Institute for Health and Social Affairs (KIHASA) for providing the 1998 Korean National Health and Nutrition Examination Survey data. YSY, SWO, HWB, and HSP were involved in the conception and design of the study; YSY and SWO contributed to data analysis; YSY, SWO, and WYK contributed to the interpretation of data; YSY drafted the manuscript; and all authors participated in critically revising the manuscript and approved the final version of the manuscript. None of the authors had a conflict of interest in any company or organization sponsoring this study.

REFERENCES

8. Agarwal DP. Cardioprotective effects of light-moderate consumption of