

# Relationship Among Alcohol, Body Weight, and Cardiovascular Risk Factors in 27,030 Korean Men

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**OBJECTIVE** — Recent studies suggest a lower risk for overweight/obesity in moderate alcohol drinkers. However, the validity of this relationship and its impact on the putative benefits of alcohol consumption on cardiovascular disease (CVD) risk has not been well evaluated.

**RESEARCH DESIGN AND METHODS** — We assessed the impact of BMI on the relationship between alcohol consumption and CVD risk factors (blood pressure, lipid panel, and glucose and insulin concentrations) in 27,030 healthy Korean men with no major comorbidities or medication intake seen in a large urban Korean hospital.

**RESULTS** — BMI and overweight prevalence increased linearly with alcohol intake ( $P < 0.001$ ). Alcohol intake was also positively associated with blood pressure and triglyceride, HDL, and fasting glucose concentrations ( $P < 0.001$ ) and negatively associated with LDL and insulin concentrations ( $P < 0.001$ ). With nondrinkers as the reference group, the odds ratio for having insulin in the top quartile also declined linearly when adjusted for age, BMI, smoking, and exercise, with the heaviest drinkers ( $>40$  g/day) having an odds ratio of 0.71 (95% CI 0.62–0.82) ( $P < 0.001$ ). The relationship between alcohol and CVD risk factors was similar in normal-weight and overweight individuals.

**CONCLUSIONS** — Alcohol intake is associated with increasing BMI and several metabolic abnormalities, including higher fasting glucose. Paradoxically, it is also associated with lower insulin concentrations. The clinical significance of these findings needs further investigation.

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Although there are observational studies (1–4) showing an association between moderate alcohol consumption and decreased risk for cardiovascular disease (CVD), ethical issues have precluded a prospective study demonstrating that moderate alcohol consumption can decrease CVD. Consequently, it remains unclear whether moderate alcohol consumption actually lowers CVD risk or merely clusters with a favorable CVD risk profile.

This issue is highlighted by a recent study that found 27 of 30 CVD-associated

risk factors to be less prevalent among moderate drinkers than nondrinkers (5). BMI was one of these CVD risk factors, and it appeared that moderate drinkers were at lower risk for being overweight or obese, a finding consistent with the results of previous studies (6–9).

Overweight/obese status, itself, is associated with a number of abnormalities that increase CVD risk, including insulin resistance (10), and there are reports (6–8) that moderate drinkers tend to be more insulin sensitive. Therefore, a majority of alcohol's beneficial effects may be

related to its association with a lower BMI. Although many studies attempt to adjust for differences in BMI, we are unaware of any that have tried to systematically evaluate differences in CVD risk factors by BMI status and alcohol consumption. The purpose of this study was to directly address this issue by defining the impact that differences in body weight have on the relationship between alcohol consumption and CVD risk factors, especially insulin resistance, in a unique cohort of 27,030 healthy Korean men.

## RESEARCH DESIGN AND METHODS

We reviewed the electronic medical records of Korean male subjects aged  $\geq 19$  years (legal drinking age in South Korea) who underwent a general health status evaluation between 1 January 2005 and 30 September 2005 at Kangbuk Samsung Hospital located in Seoul, Korea. Initially, 31,593 male subjects were identified. We excluded individuals if they had history of diabetes ( $n = 527$ ), fasting plasma glucose  $\geq 126$  mg/dl ( $n = 1,184$ ), history of malignancy ( $n = 80$ ), regular medication use ( $n = 1,064$ ), positive hepatitis B surface antigen ( $n = 1,247$ ), and/or positive hepatitis C antibody ( $n = 46$ ). Other exclusions included missing either height or weight information ( $n = 22$ ) and incomplete alcohol intake history ( $n = 477$ ). As some individuals met more than one exclusion criteria, 27,030 individuals were available. The institutional review board of Kangbuk Samsung Hospital approved the study.

The health status evaluation consisted of a full medical history and examination and comprehensive blood test evaluation. From available information, we collected the following CVD risk factors to assess their association with alcohol intake: demographics (age and sex), anthropometric measurements (height and weight), blood pressure (both systolic and diastolic), regular participation in exercise, and current smoking history. BMI was calculated as weight in kilograms divided by the square of height in meters ( $\text{kg}/\text{m}^2$ ). Based on BMI, individuals were categorized as normal weight ( $\text{BMI} < 25$   $\text{kg}/\text{m}^2$ ) or overweight ( $\text{BMI} \geq 25$   $\text{kg}/\text{m}^2$ ).

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**Abbreviations:** CVD, cardiovascular disease.

A table elsewhere in this issue shows conventional and Système International (SI) units and conversion factors for many substances.

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Table 1—Clinical characteristics of 27,030 healthy Korean men according to alcohol intake (g/day)

	Group 1: 0 g/day	Group 2: 1–10 g/day	Group 3: 11–20 g/day	Group 4: 21–40 g/day	Group 5: >40 g/day	P*
n	4,512	9,830	6,059	4,854	1,775	—
Age (years)	42 ± 10	39 ± 8	40 ± 7	41 ± 7	43 ± 8	<0.001
BMI (kg/m <sup>2</sup> )	24.0 ± 3	24.0 ± 3	24.4 ± 3	24.7 ± 3	24.8 ± 3	<0.001
Overweight (%) BMI ≥25 kg/m <sup>2</sup>	35	34	40	44	46	<0.001
Current smoker (%)	29	37	47	54	61	<0.001

Data are means ± SD, unless otherwise indicated. \*P value is from one-way ANOVA (age, BMI) or  $\chi^2$  test (overweight, current smoker).

Obese (BMI >30 kg/m<sup>2</sup>) individuals were not analyzed separately, as they comprised <3% of the population.

Regular exercise and alcohol intake history was assessed by physician-administered questionnaire. Individuals were asked if they had participated in any regular exercise at least once a week. Alcohol intake was assessed in two ways: 1) frequency of intake (none, two to three times per month, one to two times per week, three to four times per week, or everyday) and 2) quantity of intake when drinking in reference to Soju, a popular Korean alcoholic beverage with ~65 g of alcohol per bottle (half bottle, one bottle, one and a half bottles, or two bottles). Final alcohol intake was calculated by multiplying frequency by amount, which yielded 15 groups. To simplify the groupings, we categorized individuals based on their alcohol consumption per day (grams/day): group 1 = 0, group 2 = 1–10, group 3 = 11–20, group 4 = 21–40, and group 5 = >40. Group 5 included individuals who, on average, drank 49, 66, 98, and 131 g/day, with majority drinking 49 g/day (53%). Lifetime nondrinkers were not differentiated from past drinkers.

Other CVD risk factors included lipid panel and glucose and insulin concentrations after at least 12 h of fasting. Glucose was measured using the hexokinase method (Advia 1650 Autoanalyzer; Bayer Diagnostics, Leverkusen, Germany). Insulin was measured with an immunoradiometric assay (Biosource, Nivelles, Belgium) with an intra- and interassay coefficient of variation of 2.1–4.5% and 4.7–12.2%, respectively. An enzymatic calorimetric test was used to measure total cholesterol and triglyceride concentrations. The selective inhibition method was used to measure HDL concentration, and a homogeneous enzymatic calorimetric test was used to measure LDL concentration (Advia 1650 Autoanalyzer).

To further evaluate the impact of

body weight on the relationship between alcohol intake and CVD risk, we assessed the prevalence of insulin concentration in the top quartile as another surrogate measure of insulin resistance besides fasting insulin concentrations. Insulin resistance has been positively associated with obesity (10) and inversely associated with alcohol intake (11); therefore, this factor, in particular, may vary depending on BMI and alcohol drinking status.

#### Statistical analysis

All statistical analysis was performed using SPSS (version 12 for Windows; SPSS, Chicago, IL). Clinical characteristics are reported as means ± SD, unless otherwise noted. Differences between the five alcohol groups were assessed by one-way ANOVA for continuous variables and  $\chi^2$  test for categorical variables. Post hoc Bonferroni pairwise comparisons were performed when continuous variables were significantly different ( $P < 0.05$ ). CVD risk factors were adjusted for age and stratified by weight status (BMI <25 or ≥25 kg/m<sup>2</sup>) and alcohol intake. A general linear model was used to assess trend of various CVD risk factors by alcohol intake. We also performed a linear regression analysis to evaluate interaction between BMI and alcohol consumption in predicting CVD risk factors. A logistic regression analysis was used to estimate the odds ratio and 95% CI of having insulin concentrations in the top quartile, adjusted by age, BMI, current smoking, and exercise status. Individuals who drank no alcohol were set as the reference group.

**RESULTS**— Table 1 shows general characteristics of the study population stratified into five groups by alcohol intake. Group 2 was the youngest and group 5 the oldest, and although there was a significant pairwise difference, the mean difference between the two extremes was only 4 years. BMI and overweight prevalence also tended to increase with amount of alcohol consumed, and

46% of those who consumed the most alcohol (group 5) had a BMI ≥25.0 kg/m<sup>2</sup>. Smoking prevalence also differed between the five alcohol groups and increased linearly with alcohol intake, with 29% current smokers in group 1 and 61% in group 5.

The age-adjusted relationship between alcohol intake, weight, and CVD risk factors is shown in Table 2. All CVD risk factors were worse in the overweight compared with the normal-weight individuals ( $P < 0.001$  for all comparisons between normal-weight and overweight groups). However, despite the adverse effect of being overweight on the CVD risk factors measured, general trends across alcohol groups were similar between normal-weight and overweight individuals. Thus, in both weight groups, BMI, systolic and diastolic blood pressure, triglycerides, HDL, and fasting glucose increased linearly with alcohol intake. In contrast, LDL and fasting plasma insulin concentrations decreased with alcohol intake. The decrease in insulin, however, was not continuous, reaching a nadir by group 3 in both the normal-weight and overweight individuals.

To further evaluate the relationship among alcohol, BMI, and CVD risk factors, we also checked for the interaction between alcohol groups and BMI in predicting any of the CVD risk factors. Despite the large sample size, only triglyceride concentration showed a significant interaction with alcohol and BMI ( $P = 0.004$ ). However, the curves relating alcohol groups and triglyceride concentrations were nearly parallel between the normal-weight and overweight groups (online appendix Fig. 1 [available at <http://dx.doi.org/10.2337/dc07-0315>]); only in group 5 was there a slight convergence of the curves between the two weight groups. Therefore, the interaction among triglycerides, alcohol, and BMI is unlikely to be meaningful and is only detected due to our large sample size.

To better understand the effect of BMI

Table 2—Age-adjusted CVD risk factors by weight status and alcohol intake

	Group 1: 0 g/day	Group 2: 1–10 g/day	Group 3: 11–20 g/day	Group 4: 21–40 g/day	Group 5: >40 g/day	P for trend
BMI (kg/m <sup>2</sup> )						
BMI <25 kg/m <sup>2</sup>	22.4 (22.3–22.5)	22.5 (22.5–22.5)	22.7 (22.6–22.7)	22.9 (22.8–22.9)	22.8 (22.7–22.9)	<0.001
BMI ≥25 kg/m <sup>2</sup>	27.0 (27.0–27.1)	26.9 (26.9–27.0)	26.9 (26.9–27.0)	27.0 (27.0–27.1)	27.2 (27.1–27.3)	<0.001
Systolic blood pressure (mmHg)						
BMI <25 kg/m <sup>2</sup>	113 (112.6–113.5)	113 (113.1–113.7)	114 (113.8–114.7)	116 (115.2–116.2)	117 (116.6–118.2)	<0.001
BMI ≥25 kg/m <sup>2</sup>	118 (117.3–118.7)	118 (117.9–118.9)	120 (119.1–120.2)	121 (120.7–121.9)	123 (121.6–123.5)	<0.001
Diastolic blood pressure (mmHg)						
BMI <25 kg/m <sup>2</sup>	76 (75.4–76.0)	76 (75.8–76.2)	77 (77.0–77.5)	78 (77.8–78.5)	80 (79.1–80.2)	<0.001
BMI ≥25 kg/m <sup>2</sup>	79 (78.8–79.7)	80 (79.4–80.1)	81 (80.9–81.6)	82 (81.9–82.7)	83 (82.4–83.7)	<0.001
Triglycerides (mmol/l)						
BMI <25 kg/m <sup>2</sup>	1.39 (1.36–1.42)	1.42 (1.41–1.45)	1.47 (1.44–1.50)	1.58 (1.55–1.62)	1.80 (1.74–1.85)	<0.001
BMI ≥25 kg/m <sup>2</sup>	1.84 (1.79–1.90)	1.88 (1.83–1.91)	2.00 (1.95–2.05)	2.14 (2.09–2.19)	2.24 (2.16–2.32)	<0.001
HDL (mmol/l)						
BMI <25 kg/m <sup>2</sup>	1.27 (1.25–1.27)	1.30 (1.29–1.31)	1.35 (1.33–1.35)	1.37 (1.36–1.38)	1.42 (1.40–1.43)	<0.001
BMI ≥25 kg/m <sup>2</sup>	1.17 (1.14–1.17)	1.19 (1.18–1.20)	1.22 (1.21–1.23)	1.24 (1.24–1.26)	1.30 (1.29–1.32)	<0.001
LDL (mmol/l)						
BMI <25 kg/m <sup>2</sup>	2.93 (2.89–2.94)	2.87 (2.86–2.90)	2.82 (2.80–2.85)	2.80 (2.77–2.82)	2.69 (2.65–2.74)	<0.001
BMI ≥25 kg/m <sup>2</sup>	3.13 (3.10–3.17)	3.11 (3.09–3.14)	3.06 (3.03–3.09)	3.06 (3.01–3.07)	2.95 (2.90–3.00)	<0.001
Glucose (mmol/l)						
BMI <25 kg/m <sup>2</sup>	5.22 (5.21–5.23)	5.24 (5.23–5.24)	5.27 (5.26–5.28)	5.33 (5.31–5.34)	5.39 (5.37–5.42)	<0.001
BMI ≥25 kg/m <sup>2</sup>	5.36 (5.34–5.38)	5.39 (5.37–5.41)	5.42 (5.41–5.44)	5.49 (5.47–5.51)	5.54 (5.51–5.57)	<0.001
Insulin (pmol/l)						
BMI <25 kg/m <sup>2</sup>	55 (54–56)	53 (53–54)	52 (51–53)	53 (52–53)	52 (51–53)	<0.001
BMI ≥25 kg/m <sup>2</sup>	74 (73–76)	72 (70–72)	70 (69–72)	70 (69–72)	71 (69–72)	<0.001

Data are means (95% CI).

on the relationship between alcohol intake and insulin concentration, we evaluated the likelihood (odds ratio) of an individual in groups 2–5 being in the upper quartile of fasting plasma insulin concentration compared with nondrinkers. The results of this analysis are shown in Fig. 1. When adjusted for age, smoking, and exercise, the odds ratio of having insulin in this top quartile was significantly ( $P \leq 0.01$ ) lower in groups 2 and 3 but not 4 and 5. However, when also adjusted for BMI, the odds ratio is significantly ( $P \leq 0.02$ ) lower in all the drinking groups (2–5) when compared with nondrinkers and declines progressively with increase in alcohol intake except for similar odds ratio values between groups 3 and 4. We further analyzed the relationship among alcohol, body weight, and CVD risk factors by using alcohol frequency (none, two to three times per month, one to two times per week, three to four times per week, or everyday) as groups 1–5. There were no major differences in the results (not shown).

**CONCLUSIONS**— The following conclusions can be drawn regarding the

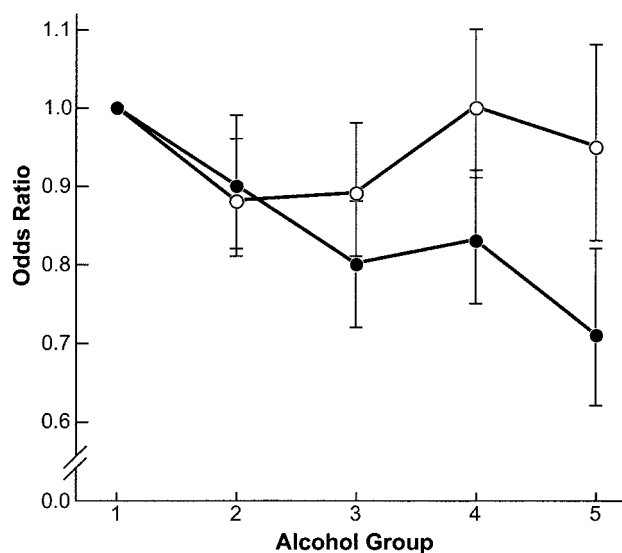
relationship among alcohol consumption, weight, and CVD risk factors in a large group of apparently healthy Korean men. 1) CVD risk factors are uniformly worse in overweight individuals, regardless of drinking status. 2) BMI and prevalence of obesity tend to increase in parallel to the history of alcohol consumption. 3) The increase in BMI underestimates the negative linear relationship between the amount of alcohol consumed and fasting plasma insulin concentrations. 4) The relationship between alcohol and CVD risk factors is similar in normal-weight and overweight individuals.

Starting with the first conclusion, it is not surprising that all CVD risk factors are worse in overweight individuals. As a group, overweight individuals have higher prevalence of metabolic disturbances, including insulin resistance, dyslipidemia, glucose intolerance, and hypertension (12). Our study suggests that all of these abnormalities become worse with increasing alcohol consumption. In addition, the benefits of alcohol cannot overcome the negative contribution of body weight. For example, the

overweight individuals in the highest drinking group (group 5) still have greater insulin concentrations than the nondrinking normal-weight individuals. Only HDL is higher in the overweight individuals in group 5 compared with normal-weight nondrinkers.

Given these findings, it is worrisome that alcohol consumption is also associated with higher BMI and higher overweight prevalence, which may further worsen the metabolic effects of alcohol consumption. However, other studies (5,6,8,9,13) have concluded that moderate alcohol drinkers have a lower BMI and lower prevalence of overweight/obesity than nondrinkers or mild drinkers. In fact, one study (9) suggested that moderate alcohol consumption may be used to control body weight.

However, it is important to note that moderate drinkers in these studies tended to have better overall health (5,6,9), less ischemic heart disease (8), and less diabetes (13). While it is certainly possible that moderate alcohol consumption is contributing to these benefits, it is also possible that healthier people drink more



**Figure 1**—Odds ratio (95% CI) of having insulin concentrations in the top quartile according to alcohol consumption in 27,030 healthy Korean men. Data are adjusted for age, current smoking, and exercise (○) as well as BMI (●).

alcohol than sicker individuals; likewise, healthier people may be thinner, and this difference could also be contributing to the putative health benefits of moderate alcohol consumption. In this regard, our study appears to be unique in both its size and the fact that the study population was generally healthy, with no known major comorbidities, including CVD and diabetes, and not taking any medications that could have influenced our findings; all of these factors may have permitted us to provide an evaluation of the health-related impact of different amounts of alcohol consumption without major confounders.

An observation of particular interest was the change in fasting plasma insulin concentration with alcohol intake, which was modified by the increases in BMI and overweight prevalence associated with higher alcohol intake. There is substantial evidence that fasting plasma insulin concentration and BMI are significantly correlated (12), and the impact of this association on the relationship between alcohol intake and insulin concentration is clearly shown in Fig. 1. These data demonstrate that the odds ratio for having insulin in the top 25% is significantly lower in groups 2 and 3 but not 4 and 5, when adjusted for differences in age, smoking, and exercise. However, when BMI is added as a covariate, the odds ratio declines linearly with higher alcohol intake. Other studies (7,14) have reported a U-shaped relationship between alcohol consumption and fasting plasma insulin concentration, which persisted even after

adjusting for BMI. In these studies, insulin nadirs occurred in individuals drinking between 10 and 30 g of alcohol/day, which is equivalent to our groups 3 and 4. In our group 5, however, we found a continued decrease in the odds ratio for having insulin in the top quartile and not an increase. These results are consistent with other studies that have found a continuous negative linear relationship between the amount of alcohol consumed and fasting insulin concentrations (6,8,11,15).

The decrease in fasting insulin is often interpreted to mean an increase in insulin sensitivity. The notion that alcohol can improve insulin sensitivity is somewhat surprising given the other abnormalities associated with alcohol, including increase in glucose, triglycerides, and blood pressure, which are all abnormalities associated with insulin resistance (12). Only two small ( $n \leq 34$ ) experimental studies (16,17) have evaluated whether moderate alcohol consumption can affect insulin sensitivity using a direct measure of insulin-mediated glucose uptake. Neither showed a significant change in insulin sensitivity in nondiabetic individuals, but further studies with larger populations may be needed to understand the relationship between alcohol consumption and insulin sensitivity.

Two other associations between amount of alcohol consumed and metabolic changes are worthy of elaboration: the decrease in plasma LDL and increase in glucose concentrations. These findings were somewhat unexpected, as they are

not associations that have received much attention compared with the increase in blood pressure and concentrations of triglycerides and HDL reported to occur with increased alcohol consumption (18,19). For instance, a study (4) that included individuals with CVD did not discern a relationship between LDL and alcohol consumption, perhaps because a significant number of patients were taking cholesterol-lowering drugs. In support of this notion, and consistent with our findings, are studies (20,21) that excluded individuals with CVD and found a modest, but statistically significant, inverse relationship between amount of alcohol consumed and LDL concentration.

The relationship between alcohol consumption and glucose concentration are more complex, and results of studies evaluating this issue have been mixed. A recent meta-analysis (22) has suggested a U-shaped relationship between alcohol and type 2 diabetes, with moderate drinkers having the lowest risk. A majority of the studies that comprised the meta-analysis (22 of 25) were prospective, but none of them enrolled a cohort without any major medical diseases such as in our population. In contrast, cross-sectional evaluations of healthier population have reported higher fasting glucose concentrations and greater risk of impaired fasting glucose and diabetes associated with alcohol consumption (11,23,24). Therefore, it is not entirely clear how different levels of alcohol consumption affect glucose homeostasis.

Our results may not be generalizable to non-Asian populations. Koreans and other East Asians have more sensitivity to alcohol, mainly due to genetic differences in alcohol-metabolizing enzymes (25); this may influence the relationship between alcohol intake and CVD risk factors. The few studies that have investigated this issue have produced mixed results, with no effect on hypertension (26,27) and a lesser effect on HDL elevation (28) in individuals with deficiency in aldehyde dehydrogenase type 2, the major enzyme affecting alcohol sensitivity. It is difficult to make any meaningful conclusions from these results, but non-Asian populations are likely to have similar, or perhaps greater, effects (at least on HDL) from alcohol intake than seen in this study.

In conclusion, this study highlights the complexities involved in understanding the effects of alcohol consumption and body weight. Increased levels of alcohol intake tended to increase BMI and



overweight prevalence. However, the relationship between alcohol and CVD risk factors was similar in the normal-weight and overweight groups. Therefore, in both groups, alcohol intake was positively associated with blood pressure and triglyceride, HDL, and glucose concentrations and negatively associated with LDL and insulin concentrations.

While all of these relationships were highly significant ( $P < 0.001$ ) using a large study population, they may not all be clinically relevant. For example, it is unclear whether a 0.18 to 0.24 mmol/l (7 to 9 mg/dl) change in LDL between groups 1 and 5 is meaningful, while a change of 0.13–0.15 mmol/l (5–6 mg/dl) in HDL and 0.4 mmol/l (35 mg/dl) in triglycerides is equivalent to that seen with cholesterol-lowering medications (29). Likewise, the clinical significance of alcohol's effect on insulin and glucose concentrations requires further investigation. It is, however, clear that there is no compelling evidence at this time for health care professionals to recommend that nondrinkers begin consuming alcohol for medical reasons.

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