Alcohol Consumption and the Prevalence of the Metabolic Syndrome in the U.S.

A cross-sectional analysis of data from the Third National Health and Nutrition Examination Survey

MATTHEW S. FREIBERG, MD[1]
HOWARD J. CABRAL, PHD, MPH[2]
TIM C. HEEREN, PHD[2]
RAMACHANDRAN S. VASAN, MD[3,4]
R. CURTIS ELLISON, MD[3]

OBJECTIVE — The aim of this study was to examine the relations of alcohol consumption to the prevalence of the metabolic syndrome and its components in the U.S. population.

RESEARCH DESIGN AND METHODS — We performed a cross-sectional analysis on data from 8,125 participants from the Third National Health and Nutrition Examination Survey who were evaluated for each component of the metabolic syndrome, using the National Cholesterol Education Program criteria, fasting insulin, and alcohol consumption. Current alcohol consumption was defined as ≥1 alcoholic drink per month.

RESULTS — After adjustment for age, sex, race/ethnicity, education, income, tobacco use, physical activity, and diet, subjects who consumed 1–19 and ≥20 drinks of alcohol per month had odds ratios (ORs) for the prevalence of the metabolic syndrome of 0.65 and 0.34, respectively ($P < 0.05$ for all), compared with current nondrinkers. These findings were particularly noteworthy for beer and wine drinkers. The association of ≥20 alcoholic drinks per month with the prevalence of the metabolic syndrome was consistent across ethnicities but was most striking in white men and women (ORs 0.35 and 0.22, respectively; $P < 0.05$). Alcohol consumption was significantly and inversely associated with the prevalence of the following three components of the metabolic syndrome: low serum HDL cholesterol, elevated serum triglycerides, high waist circumference, as well as hyperinsulinemia ($P < 0.05$ for all).

CONCLUSIONS — Mild to moderate alcohol consumption is associated with a lower prevalence of the metabolic syndrome, with a favorable influence on lipids, waist circumference, and fasting insulin. This association was strongest among whites and among beer and wine drinkers.

Diabetes Care 27:2954–2959, 2004

Light to moderate alcohol consumption is associated with lower cardiovascular mortality (1) and a reduced risk of developing type 2 diabetes (2). Some of the biological mechanisms reported to explain this observation include an improvement of the lipid profile, especially HDL cholesterol (3) and increasing insulin sensitivity (4,5).

The metabolic syndrome is a clustering of low serum HDL cholesterol, elevated serum triglycerides, hyperglycemia, central obesity, and elevated blood pressure, mediated in part by insulin resistance (6,7). The metabolic syndrome is associated with an increased risk of developing diabetes (8) and cardiovascular disease (9).

The favorable influence of alcohol consumption on select components of the metabolic syndrome (3,10) raises the possibility that alcohol intake may reduce the risk of the metabolic syndrome. Few studies have examined the association between alcohol consumption and the metabolic syndrome as defined by the National Cholesterol Education Program (11–13), and data are limited on how the relation may be modified by type of alcohol, sex, or race/ethnicity (14,15).

We investigated the relations of the quantity and the type of alcohol consumed to the prevalence of the metabolic syndrome (and its components) in men and women of different race/ethnicities in the U.S. population.

RESEARCH DESIGN AND METHODS

— We analyzed data on 8,125 individuals from the Third National Health and Nutrition Examination Survey (NHANES III) (1988–1994); the details of the study design and sampling and examination techniques have been described elsewhere (16,17). Individuals participating in NHANES III were considered eligible for the present study if they were age 20 years or older, not pregnant, had completed the adult household interview and questionnaire, had a physical examination and laboratory evaluation performed at one of the examination centers, and had fasted for at least 8 hours before phlebotomy. Furthermore, they were required to have data on *1*) abdominal waist circumference, *2*) fasting serum

From the [1]Section of General Internal Medicine, Boston University School of Medicine, Boston, Massachusetts; the [2]Department of Biostatistics, Boston University School of Public Health, Boston, Massachusetts; the [3]Section of Preventive Medicine and Epidemiology, Boston University School of Medicine, Boston, Massachusetts; and the [4]National Heart, Lung & Blood Institute's Framingham Heart Study, Framingham, Massachusetts.

Address correspondence and reprint requests to Matthew S. Freiberg, MD, Boston Medical Center, 91 E. Concord St., MAT 2 Suite 200, Boston, MA 02118. E-mail: matthew.freiberg@bmc.org.

Received for publication 26 July 2004 and accepted in revised form 26 August 2004.

R.C.E. serves on an advisory panel that oversees a major study funded by the National Institute on Alcohol Abuse and Alcoholism and has received grant/financial support for research from companies/organizations that have some relation to the wine or beverage alcohol industry.

Abbreviations: NHANES III, Third National Health and Nutrition Examination Survey.

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

triglyceride, 3) blood pressure, 4) fasting serum HDL cholesterol, 5) fasting serum glucose, 6) fasting serum insulin, and 7) each of the covariates detailed below.

Definitions of outcome, exposure, and covariates

Outcome. We defined the metabolic syndrome using the National Cholesterol Education Program definition (6). This definition was satisfied if a subject possessed three or more of the following five criteria: an abdominal waist circumference >102 cm in men or >88 cm in women; serum triglycerides ≥150 mg/dl (≥1.69 mmol/l); serum HDL cholesterol <40 mg/dl (<1.04mmol/l) in men or <50 mg/dl (<1.29 mmol/l) in women; average blood pressure ≥130/85 mmHg or currently taking antihypertensive medication; or fasting serum glucose ≥110 mg/dl (≥6.1 mmol/l) or on oral antidiabetic medication. We averaged the second and third systolic and diastolic blood pressure readings for each subject. Subjects were also classified as hyperinsulinemic if the fasting insulin level was ≥90th percentile of its distribution among subjects in the sample (this value corresponded to a fasting insulin ≥166 pmol/l). This percentile was selected based on work by Meigs et al. (18).

Exposure: alcohol intake (type and amount). In NHANES III, alcohol consumption was assessed by asking each participant to quantify the number of drinks consumed in the past month before the survey of three different types of alcohol: 1) beer or light beer; 2) wine, wine coolers, sangria, and champagne; and 3) hard liquor such as tequila, gin, vodka, scotch, rum, whiskey, and liqueurs, either alone or mixed. We defined alcohol consumption as 1) currently drinking (≥1 drink of any type per month) or not currently drinking (<1 per month); 2) the number per month of any type of alcoholic drinks (<1, 1–19, or ≥20); and 3) the number of beverage-specific drinks per month (<1, 1–19, or ≥20). Further categorization of the alcohol variable was not possible due to inadequate numbers. The not currently drinking category (<1 drink per month) included subjects consuming at least 12 drinks of any type over a subject's lifetime but not currently drinking alcohol (past drinkers) and subjects consuming <12 drinks in their entire lifetime (lifetime abstainers). Types of alcohol consumed were divided into three categories: beer, wine, and liquor.

Covariates. The definitions of the covariates used in this study were based on metabolic syndrome risk factors described by Park et al. (11). Race/ethnicity was based on the screener and subject self-report. We used three categories: non-Hispanic white (white), non-Hispanic black (black), and Mexican American. Other racial/ethnic groups were not included in our analysis because of inadequate numbers.

Level of education was categorized into <8th grade, between 8th and 12th grade, and >12th grade education. Annual household income from the previous year was classified as <$20,000 or ≥$20,000. Subjects were considered physically inactive if they expended <3.5 metabolic equivalents/month participating in walking, jogging, running, bicycle riding, swimming, aerobics, aerobic dancing, other types of dancing, calisthenics, exercises, or garden work. Tobacco use was divided into current user, past user (≥100 cigarettes in their lifetime but currently not smoking), or never smoker. The percentage of daily total energy intake from carbohydrates (kilocalories) was calculated and divided into <40, 40–60, or >60%. Adequate data on hormone replacement therapy were not available; we did, however, use postmenopausal status as a covariate because it has been shown to be a risk factor for the metabolic syndrome (11). Women were considered postmenopausal if they had not had a menstrual period in the past 12 months. Age was used as continuous variable as well as categorized into categories of 20–29, 30–39, 40–49, 50–59, 60–69, and ≥70 years of age.

Statistical analysis

For each sex and race/ethnicity, we calculated the prevalences of the metabolic syndrome, of each component of the syndrome, and of all covariates. Multiple logistic regression analyses were used to estimate the ORs for the prevalence of the metabolic syndrome and each of its components using all three definitions of alcohol consumption as independent variables (separate analyses for each) while adjusting for covariates. For race/ethnicity-specific models, interaction was assessed using an interaction term between race/ethnicity and the number of alcoholic drinks per month for men and women. Because BMI was highly correlated with waist circumference ($r = 0.87$, $P < 0.0001$), which was one of the constituent criteria for our dependent variable, we felt BMI served more as a proxy than a predictor for waist circumference. We did not, therefore, include BMI as a covariate in our primary analyses. We did perform secondary analyses with BMI as a covariate, and the inclusion of BMI did not alter the relationship between alcohol and the metabolic syndrome (data not shown).

All analyses used sampling weights to report estimates that would be representative of the U.S. population. We used SUDAAN statistical software (19) to obtain proper variance estimates to calculate weighted means, percentages, standard errors, and ORs given the NHANES III complex sampling design. A two-sided P value of <0.05 was considered statistically significant. The institutional review board at Boston University Medical Center approved this study.

RESULTS

The characteristics of our sample are presented in Table 1. Over one-half (57.9%) of the participants were current drinkers with higher percentages for men (66.0%) than women (50.0%). The prevalence of the metabolic syndrome was slightly higher in women (22.7%) than in men (21.9%). Mexican-American women had the highest proportion of participants with the metabolic syndrome (26.9%) and the smallest proportion of current drinkers (36.0%).

Current drinkers had a lower adjusted prevalence of the metabolic syndrome than subjects not currently drinking (OR 0.57 [95% CI 0.45–0.72]). Compared with lifetime abstainers, the adjusted odds for the prevalence of the metabolic syndrome among past drinkers was 0.96 (0.75–1.22). The reduced odds for the prevalence of the metabolic syndrome in current drinkers were statistically significant in all age-groups except the 50–60 age-group (0.76 [0.46–1.26]) and the ≥70 age-group (0.72 [0.52–1.00]). The inverse relation of alcohol consumption to prevalence of the metabolic syndrome was consistent in men and in women (0.58 [0.44–0.77] and 0.57 [0.44–0.74], respectively).

Overall, subjects consuming higher quantities of alcoholic beverages had a lower OR for the prevalence of the metabolic syndrome as compared with current

Table 1—*Characteristics of study population†**

Characteristics of study population	Men	Women
n	3,951	4,174
Mean age in years	42.7 ± 0.5	45.2 ± 0.6
Race/ethnicity		
White	83.4 ± 1.0	83.0 ± 0.9
Black	10.4 ± 0.7	12.0 ± 0.8
Mexican American	6.3 ± 0.6	5 ± 0.4
Education		
<8th grade	6.1 ± 0.6	5.5 ± 0.5
8th–12th grade	49.1 ± 1.4	55.0 ± 1.3
>12th grade	44.8 ± 1.6	39.5 ± 1.4
Annual family income <$20,000 per year	28.6 ± 1.4	36.1 ± 1.4
Physical activity <3.5 Mets of physical activity per month	12.0 ± 1.1	18.4 ± 1.2
Tobacco use		
Current	31.5 ± 1.3	25.9 ± 1.1
Past	32.3 ± 1.2	21.9 ± 0.9
Never	36.2 ± 1.3	52.3 ± 1.3
Diet (% kcal from carbohydrate)		
<40%	25.1 ± 1.7	18.2 ± 1.5
40–60%	62.1 ± 1.4	63.0 ± 1.6
>60%	12.9 ± 0.9	18.9 ± 1.2
Currently postmenopausal	—	40.1 ± 1.6
Number of alcoholic drinks per month		
<1	34.1 ± 1.7	50.0 ± 1.7
1–19	45.5 ± 1.2	41.2 ± 1.4
≥20	20.5 ± 1.3	8.7 ± 0.8
Number of beer drinks per month		
<1 drink/month	41.8 ± 1.9	71.5 ± 1.6
1–19 drinks/month	45.6 ± 1.7	22.1 ± 1.6
≥20 drinks/month	12.8 ± 0.9	2.5 ± 0.4
Number of wine drinks per month		
<1 drink/month	75.5 ± 1.5	69.6 ± 1.6
1–19 drinks/month	23.0 ± 1.4	28.4 ± 1.5
≥20 drinks/month	1.6 ± 0.3	2.1 ± 0.3
Number of liquor drinks per month		
<1 drink/month	66.0 ± 1.5	74.0 ± 1.5
1–19 drinks/month	30.0 ± 1.5	23.8 ± 1.3
≥20 drinks/month	4.0 ± 0.4	2.1 ± 0.4
Waist circumference >102 cm (men) or >88 cm (women)	27.6 ± 1.0	45.3 ± 1.2
Serum triglycerides ≥1.69 mmol/l	33.6 ± 1.8	23.7 ± 0.9
Serum HDL cholesterol <1.04 mmol/l (men) or <1.29 mmol (women)	34.5 ± 1.6	39.1 ± 1.6
Blood pressure ≥130/85 mmHg	34.8 ± 1.6	28.3 ± 1.1
Fasting blood glucose ≥6.1 mmol/l	13.6 ± 0.8	9.2 ± 0.6
Fasting insulin ≥166 pmol/l	7.9 ± 0.8	7.1 ± 0.6
Prevalence of the metabolic syndrome	21.9 ± 1.3	22.7 ± 1.0

Data are percent ± SE. *Percentages may not add to 100% due to rounding effect; †percentages were calculated using sampling weights to report estimates that would be representative of the U.S. population.

nondrinkers ($P < 0.0001$ for trend) (Table 2). Beer, wine, and liquor had different associations with the prevalence of the metabolic syndrome, with statistically significant reductions in the OR for the prevalence of the metabolic syndrome only for beer and wine.

The relation of alcohol intake to prevalence of the metabolic syndrome was stronger in whites as compared with blacks or Mexican Americans (Table 3). Furthermore, unlike among whites, in black and Mexican-American subjects, consumption of ≥20 drinks per month resulted only in a modest reduction in the OR for the metabolic syndrome, as compared with subjects who drank 1–19 drinks per month. This difference, however, was not accompanied by a statistically significant interaction term between race/ethnicity and the number of alcoholic drinks per month for men ($P = 0.37$) or women ($P = 0.13$).

Although increasing quantities of alcohol consumption were associated with a lower OR for the prevalence of the metabolic syndrome in the overall population, this association was not consistent for each of the components. Alcohol consumption was significantly associated with a lower OR for the prevalence of three components of the metabolic syndrome: low serum HDL cholesterol, elevated serum triglycerides, and high waist circumference, as well as hyperinsulinemia (Table 4). When type of alcohol was considered, beer and wine drinkers had favorable associations with low HDL cholesterol, elevated serum triglycerides, waist circumference, hyperinsulinemia, and elevated serum glucose. Among beer and wine drinkers, the odds for the prevalence of elevated serum glucose when drinking ≥20 drinks per month were 0.68 (95% CI 0.44–1.05) and 0.51 (0.28–0.90), respectively. In contrast, for liquor drinkers, the only favorable association was with low HDL cholesterol. Furthermore, among liquor drinkers, the odds for the prevalence of elevated serum glucose when drinking ≥20 drinks per month was 1.90 (1.25–2.88).

CONCLUSIONS — Overall, our results demonstrate that people who drink alcoholic beverages have a lower prevalence of the metabolic syndrome as compared with current nondrinkers after accounting for confounders. The inverse relation of alcohol consumption and the metabolic syndrome was especially noticeable in people who consume ≥20 alcoholic beverages per month and was stronger when the beverage was beer or wine. Although the association between alcohol consumption and the metabolic syndrome was present for all race/ethnicities, it was particularly strong in white individuals.

Inconsistent results have been re-

Table 2—*Multivariable adjusted OR for the prevalence of the metabolic syndrome in 8,125 individuals from the NHANES III stratified by quantity and type of alcohol consumption*

Quantity and type of alcohol consumption	OR for the metabolic syndrome (95% CI)
Drinks consumed per month*	
<1	1.0 (referent)
1–19	0.65 (0.54–0.79)
≥20	0.34 (0.26–0.47)
Linear trend	$P = 0.0001$
Type and quantity of drinks per month†	
Beer	
<1	1.0 (referent)
1–19	0.75 (0.61–0.91)
≥20	0.31 (0.18–0.53)
Linear trend	$P = 0.006$
Wine	
<1	1.0 (referent)
1–19	0.64 (0.51–0.79)
≥20	0.28 (0.15–0.54)
Linear trend	$P < 0.0001$
Liquor	
<1	1.0 (referent)
1–19	0.95 (0.73–1.2)
≥20	0.74 (0.49–1.14)
Linear test for trend	$P = 0.40$

*Model 1 adjusted for age (as a continuous variable), sex, race/ethnicity (white, black, or Mexican American), education (<8th grade, 8–12th grade, or >12th grade), annual income (< or ≥$20,000 per year), tobacco use (current, past, or never), physical activity (< or ≥3.5 Mets of activity per month), and diet (<40, 40–60, or >60% kcal from carbohydrates). †Model 2 adjusted for all variables in model 1 and each type of alcoholic beverage (beer, wine, and liquor).

ported between alcohol and the metabolic syndrome (11,13,20). Yoon et al. (13) reported that 1–15 g of alcohol per day was associated with decreased prevalence of the metabolic syndrome, whereas Park et al. (11) reported a decrease only in women. This difference may relate to the fact that Park et al. (11) included in the "heavy" drinking category participants who had a history of "ever consuming five or more drinks almost daily" even if they were not currently drinking. Furthermore, participants were included if they fasted for ≥6 h, whereas the present study and that of Yoon et al. (13) required ≥8 and 10–12 h, respectively.

Numerous studies show serum HDL cholesterol concentrations increase in a dose-dependent response to alcohol consumption (3,21). Serum triglycerides are higher in heavy drinkers (21) but can be lower in light to moderate drinkers (22). The relation with obesity, on the other hand, has not been reported in a consistent fashion (23,24). Nevertheless, some data demonstrate that moderate amounts of alcohol, especially wine, are associated with smaller waist circumference as compared with no alcohol consumption (25,26). Although we did not observe an association for all beverages between alcohol consumption and hyperglycemia, several prospective studies have reported a reduced incidence of diabetes with alcohol consumption (27–29). Furthermore, several studies, including the present study, have demonstrated lower insulin concentrations and increased insulin sensitivity among drinkers (19,30). In contrast, only a few studies have shown a beneficial effect of alcohol consumption on blood pressure (31,32), whereas the majority of studies support our finding that alcohol consumption results in elevated blood pressure (33). The strength of the association between alcohol consumption and the metabolic syndrome may depend heavily upon the prevalence of these individual criteria in a given population.

We found that the reduced prevalence of metabolic syndrome was stronger among drinkers of wine and beer than of liquor. This could result if liquor consumers drank larger amounts or had a different pattern of consumption (e.g., binge drinking) (34,35). Indeed, our data showed that among individuals consuming ≥50 total drinks per month, the mean number of drinks of liquor consumed (18.7) was more than twice that of wine (8.1; $P = 0.001$), whereas there was no difference in the light-drinking categories.

Table 3—*Multivariable adjusted OR for the prevalence of the metabolic syndrome in 8,125 subjects from the NHANES III consuming different quantities of alcohol stratified by sex and race/ethnicity*

Quantities of alcohol consumed	Men			Women*		
	White	Black	Mexican American	White	Black	Mexican American
n	1,655	1,064	1,232	1,824	1,233	1,117
Drinks consumed per month†						
<1 (referent)	1.0	1.0	1.0	1.0	1.0	1.0
1–19	0.66 (0.47–0.93)	0.83 (0.51–1.36)	0.81 (0.60–1.09)	0.62 (0.44–0.86)	0.80 (0.53–1.21)	0.75 (0.53–1.05)
≥20	0.35 (0.22–0.55)	0.72 (0.37–1.38)	0.67 (0.42–1.08)	0.22 (0.13–0.39)	0.79 (0.28–2.27)	0.66 (0.32–1.37)
Linear test for trend	$P < 0.0001$	$P = 0.32$	$P = 0.07$	$P < 0.0001$	$P = 0.37$	$P = 0.06$

Data are OR (95% CI). *All models for female subjects contained menopausal status variable. †Model adjusted for education (<8th grade, 8–12th grade, or >12th grade), annual income (< or ≥$20,000 per year), tobacco use (current, past, or never), physical activity (< or ≥3.5 Mets of activity per month), diet (<40, 40–60, or >60% kcal from carbohydrates), and age (as a continuous variable).

Table 4—*Multivariable adjusted ORs for the prevalence of each of the criteria for the metabolic syndrome and hyperinsulinemia in 8,125 subjects from the NHANES III stratified by increasing quantities of alcohol consumption

	n	Serum HDL cholesterol <1.04 mmol/l in men or <1.29 mmol/l in women	Serum triglycerides ≥1.69 mmol/l	Waist circumference >102 cm in men or >88 cm in women	Blood pressure ≥130/85 or antihypertensive medication	Fasting blood glucose ≥6.1 mmol/l or antidiabetic medication	Fasting serum insulin ≥90th percentile (116 pmol/l)
<1 alcoholic drink per month	4,031	1.0	1.0	1.0	1.0	1.0	1.0
1–19 alcoholic drinks per month	3,068	0.69 (0.60–0.78)	0.73 (0.62–0.87)	0.74 (0.62–0.89)	1.04 (0.83–1.30)	0.80 (0.60–1.05)	0.64 (0.46–0.88)
≥20 drinks per month	1,026	0.22 (0.16–0.29)	0.56 (0.43–0.74)	0.41 (0.32–0.52)	1.12 (0.82–1.52)	0.89 (0.64–1.24)	0.39 (0.24–0.62)
Linear test for trend		$P < 0.0001$	$P = 0.0001$	$P = 0.003$	$P = 0.76$	$P = 0.11$	$P = 0.0002$

Data are OR (95% CI). *All models adjusted for age (as a continuous variable), sex, race/ethnicity (white, black, or Mexican American), education (<8th grade, 8–12th grade, or >12th grade), annual income (< or ≥$20,000 per year), tobacco use (current, past, or never), physical activity (< or ≥3.5 Mets of activity per month), and diet (<40, 40–60, or >60% kcal from carbohydrates).

However, we did not have adequate data to describe drinking patterns in detail.

Unlike whites, blacks had only a modest reduction in the odds for the prevalence of the metabolic syndrome when currently drinking, and in black women there was no further reduction in the odds with increasing quantities of alcohol. Although testing of interaction was statistically not significant, we had limited power to evaluate for effect modification by race at the highest quantities of alcohol consumption. Previous studies have reported a relation between increased alcohol consumption and hypertension (33) and a higher prevalence of hypertension in blacks (36), and our data show that blacks were heavier drinkers. Of the current drinkers in our sample, 9.3% of black men and 3.4% of black women currently consumed at least 50 drinks per month, whereas this was lower in white men (5.8%) and women (2.0%).

Differences by race/ethnicity may also relate to insulin sensitivity or hyperinsulinemia (37). Whereas hyperinsulinemia is not a criterion for the metabolic syndrome, studies suggest that it is an important element of the syndrome (7). Using factor analysis, Meigs et al. (18) concluded that a unified metabolic syndrome included three independent factors: hyperinsulinemia, obesity, and dyslipidemia. Among black nondiabetic subjects, however, insulin sensitivity is decreased as compared with white subjects (38). Similarly, fasting insulin concentrations are higher among black women as compared with white women at similar levels of adiposity (39). In the present study, we found that the odds for the prevalence of hyperinsulinemia, after adjusting for our covariates including BMI, was higher among blacks as compared with whites (1.47 [1.10–2.00]).

Although the strengths of our investigations include the use of a large sample representative of the adult U.S. population (thereby enhancing our generalizability), the evaluation of both the type and the quantity of alcohol consumed, and the assessment of effect modification by race/ethnicity, there are several limitations that merit comment. First, given the cross-sectional design, we cannot draw any causal inferences regarding the association of alcohol consumption with the metabolic syndrome. Second, the data on alcohol consumption are based on self-report with the possibility of misclassification of exposure (e.g., under reporting). However, this bias, if nondifferential, would be expected only to increase the amounts of alcohol associated with a reduced prevalence of the metabolic syndrome. Third, categories with the highest quantities of alcohol consumption had the fewest number of individuals, thus limiting our ability to comment on the relation of alcohol consumption to the prevalence of the metabolic syndrome in heavy drinkers. Finally, we cannot comment on the effects of daily patterns of alcohol consumption, as these data were not available.

In summary, mild to moderate alcohol consumption, especially of beer and wine, is associated with a lower prevalence of the metabolic syndrome and with a favorable influence on serum lipids, waist circumference, and fasting serum insulin. This observed association between alcohol consumption and the metabolic syndrome was particularly strong in white individuals.

Acknowledgments— This work was supported in part through NRSA Training Grant 2T 32 HP 100028–06 (to M.S.F.), a research career award K24 HL 04334 from the NHLBI/NIH (to R.S.V.), and a National Institutes of Health Grant AA13304-01 (to R.C.E.).

References

1. Fuchs CS, Stampfer MJ, Colditz GA, Giovannucci EL, Manson JE, Kawachi I, Hunter DJ, Hankinson SE, Hennekens CH, Rosner B: Alcohol consumption and mortality among women. *N Engl J Med* 332:1245–1250, 1995
2. Wei M, Gibbons LW, Mitchell TL, Kampert JB, Blair SN: Alcohol intake and incidence of type 2 diabetes in men. *Diabetes Care* 23:18–22, 2000
3. Gaziano JM, Buring JE, Breslow JL, Goldhaber SZ, Rosner B, VanDenburgh M, Willett W, Hennekens CH: Moderate alcohol intake, increased levels of high-density lipoprotein and its subfractions, and decreased risk of myocardial infarction. *N Engl J Med* 329:1829–1834, 1993
4. Sierksma A, Patel H, Ouchi N, Kihara S, Funahashi T, Heine RJ, Grobbee DE, Kluft C, Hendriks HF: Effect of moderate alcohol consumption on adiponectin, tumor necrosis factor-α, and insulin sensitivity. *Diabetes Care* 27:184–189, 2004
5. Davies MJ, Baer DJ, Judd JT, Brown ED, Campbell WS, Taylor PR: Effects of moderate alcohol intake on fasting insulin and glucose concentrations and insulin sensi-

tivity in postmenopausal women: a randomized controlled trial. *JAMA* 287: 2559–2562, 2002
6. National Institutes of Health: *Third Report of the National Cholesterol Education Program Expert Panel on Detection E, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III)*. Bethesda, MD, National Institutes of Health, 2001 (NIH publ. no. 01-3670)
7. Reaven GM: Banting lecture 1988: role of insulin resistance in human disease. *Diabetes* 37:1595–1607, 1988
8. Haffner SM, Valdez RA, Hazuda HP, Mitchell BD, Morales PA, Stern MP: Prospective analysis of the insulin-resistance syndrome (syndrome X). *Diabetes* 41: 715–722, 1992
9. Isomaa B, Almgren P, Tuomi T, Forsen B, Lahti K, Nissen M, Taskinen MR, Groop L: Cardiovascular morbidity and mortality associated with the metabolic syndrome. *Diabetes Care* 24:683–689, 2001
10. Criqui MH, Cowan LD, Tyroler HA, Bangdiwala S, Heiss G, Wallace RB, Cohn R: Lipoproteins as mediators for the effects of alcohol consumption and cigarette smoking on cardiovascular mortality: results form the Lipid Research Clinics Follow-Up Study. *Am J Epidemiol* 126:629–637, 1987
11. Park YW, Zhu S, Palaniappan L, Heshka S, Carnethon MR, Heymsfield SB: The metabolic syndrome: prevalence and associated risk factor findings in the U.S. population from the Third National Health and Nutrition Examination Survey, 1988–1994. *Arch Intern Med* 163: 427–436, 2003
12. Dixon JB, Dixon ME, O'Brien PE: Alcohol consumption in the severely obese: relationship with the metabolic syndrome. *Obes Res* 10:245–252, 2002
13. Yoon YS, Oh SW, Baik HW, Park HS, Kim WY: Alcohol consumption and the metabolic syndrome in Korean adults: the 1998 Korean National Health and Nutrition Examination Survey. *Am J Clin Nutr* 80:217–224, 2004
14. Rosell M, De Faire U, Hellenius ML: Low prevalence of the metabolic syndrome in wine drinkers: is it the alcohol beverage or the lifestyle? *Eur J Clin Nutr* 57:227–234, 2003
15. Goude D, Fagerberg B, Hulthe J: Alcohol consumption, the metabolic syndrome and insulin resistance in 58-year-old clinically healthy men (AIR study). *Clin Sci (Lond)* 102:345–352, 2002
16. Plan and operation of the Third National Health and Nutrition Examination Survey *Vital Health Stat 1* 32:1–407, 1994
17. Centers for Disease Control and Prevention: *The Third National Health and Nutrition Examination Survey (NHANES III 1988–94) Reference Manuals and Reports* [CD-ROM]. Bethesda, MD, MNC
18. Meigs JB, D'Agostino RB, Sr, Wilson PW, Cupples LA, Nathan DM, Singer DE: Risk variable clustering in the insulin resistance syndrome: the Framingham Offspring Study. *Diabetes* 46:1594–1600, 1997
19. Shah B, Barnwell BG, Bieler GS: *SUDAAN User's Manual: Version 7.5*. Research Triangle Park, NC, Research Triangle Institute, 1997
20. Vernay M, Balkau B, Moreau JG, Sigalas J, Chesnier MC, Ducimetiere P: Alcohol consumption and insulin resistance syndrome parameters: associations and evolutions in a longitudinal analysis of the French DESIR cohort. *Ann Epidemiol* 14: 209–214, 2004
21. Rimm EB, Williams P, Fosher K, Criqui M, Stampfer MJ: Moderate alcohol intake and lower risk of coronary heart disease: meta-analysis of effects on lipids and haemostatic factors. *BMJ* 319:1523–1528, 1999
22. Mayer EJ, Newman B, Quesenberry CP Jr, Friedman GD, Selby JV: Alcohol consumption and insulin concentrations: role of insulin in associations of alcohol intake with high-density lipoprotein cholesterol and triglycerides. *Circulation* 88:2190–2197, 1993
23. Koh-Banerjee P, Chu NF, Spiegelman D, Rosner B, Colditz G, Willett W, Rimm E: Prospective study of the association of changes in dietary intake, physical activity, alcohol consumption, and smoking with 9-y gain in waist circumference among 16,587 U.S. men. *Am J Clin Nutr* 78:719–727, 2003
24. Hellerstedt WL, Jeffery RW, Murray DM: The association between alcohol intake and adiposity in the general population. *Am J Epidemiol* 132:594–611, 1990
25. Duncan BB, Chambless LE, Schmidt MI, Folsom AR, Szklo M, Crouse JR 3rd, Carpenter MA: Association of the waist-to-hip ratio is different with wine than with beer or hard liquor consumption: Atherosclerosis Risk in Communities Study Investigators. *Am J Epidemiol* 142:1034–1038, 1995
26. Vadstrup ES, Petersen L, Sorensen TI, Gronbaek M: Waist circumference in relation to history of amount and type of alcohol: results from the Copenhagen City Heart Study. *Int J Obes Relat Metab Disord* 27:238–246, 2003
27. Rimm EB, Chan J, Stampfer MJ, Colditz GA, Willett WC: Prospective study of cigarette smoking, alcohol use, and the risk of diabetes in men. *BMJ* 310:555–559, 1995
28. Ajani UA, Hennekens CH, Spelsberg A, Manson JE: Alcohol consumption and risk of type 2 diabetes mellitus among U.S. male physicians. *Arch Intern Med* 160:1025–1030, 2000
29. Wannamethee SG, Camargo CA Jr, Manson JE, Willett WC, Rimm EB: Alcohol drinking patterns and risk of type 2 diabetes mellitus among younger women. *Arch Intern Med* 163:1329–1336, 2003
30. Facchini F, Chen YD, Reaven GM: Light-to-moderate alcohol intake is associated with enhanced insulin sensitivity. *Diabetes Care* 17:115–119, 1994
31. MacMahon S: Alcohol consumption and hypertension. *Hypertension* 9:111–121, 1987
32. Gillman MW, Cook NR, Evans DA, Rosner B, Hennekens CH: Relationship of alcohol intake with blood pressure in young adults. *Hypertension* 25:1106–1110, 1995
33. Klatsky AL: Blood pressure and alcohol intake. In *Hypertension: Pathophysiology, Diagnosis, and Management*. 2nd ed. Brenner LBM, Ed. New York, Raven Press, Ltd, 1995, p. 2649–2667
34. Tolstrup JS, Jensen MK, Tjonneland A, Overvad K, Gronbaek M: Drinking pattern and mortality in middle-aged men and women. *Addiction* 99:323–330, 2004
35. Trevisan M, Dorn J, Falkner K, Russell M, Ram M, Muti P, Freudenheim JL, Nochajaski T, Hovey K: Drinking pattern and risk of non-fatal myocardial infarction: a population-based case-control study. *Addiction* 99:313–322, 2004
36. Hajjar I, Kotchen TA: Trends in prevalence, awareness, treatment, and control of hypertension in the United States, 1988–2000. *JAMA* 290:199–206, 2003
37. He J, Klag MJ, Caballero B, Appel LJ, Charleston J, Whelton PK: Plasma insulin levels and incidence of hypertension in African Americans and whites. *Arch Intern Med* 159:498–503, 1999
38. Haffner SM, D'Agostino R, Saad MF, Rewers M, Mykkanen L, Selby J, Howard G, Savage PJ, Hamman RF, Wagenknecht LE, Bergman RN: Increased insulin resistance and insulin secretion in nondiabetic African-Americans and Hispanics compared with non-Hispanic whites: the Insulin Resistance Atherosclerosis Study. *Diabetes* 45:742–748, 1996
39. Palaniappan LP, Carnethon MR, Fortmann SP: Heterogeneity in the relationship between ethnicity, BMI, and fasting insulin. *Diabetes Care* 25:1351–1357, 2002