

Available online at www.sciencedirect.com

SciVerse ScienceDirect

Nutrition Research

Nutrition Research 32 (2012) 24-29

www.nrjournal.com

The association of alcohol consumption with metabolic syndrome and its individual components: the Taichung community health study

Ching-Chu Chen^{a, b}, Wen-Yuan Lin^{c, d}, Chia-Ing Li^e, Chiu-Shong Liu^{c, d}, Tsai-Chung Li^{f, g}, Ying-Tzu Chen^e, Chuan-Wei Yang^e, Man-Ping Chang^h, Cheng-Chieh Lin^{c, d, g, i,*}

Abstract

Alcohol has both adverse and protective effects on the individual components of metabolic syndrome (MS). We hypothesize that alcohol consumption increases the risk of developing MS and that the consumption of different types of alcoholic beverages has different effects on the development of MS and its individual components. We enrolled 2358 men for this cross-sectional study. The data were collected from self-reported nutrition and lifestyle questionnaires. Individuals who drank at least once per week for 6 consecutive months were classified as current drinkers. Current drinkers were at a higher risk of developing MS, abdominal obesity, and high triglyceride levels, but they were at a lower risk of developing low levels of high-density lipoprotein cholesterol (HDL-C). The increased risk of developing MS, high triglyceride, and high fasting glucose levels was dose dependent, whereas low HDL-C levels demonstrated a reverse relationship. The dose needed to reduce the risk of having low HDL-C levels was ≥50 g/d. This dose, however, resulted in an increased risk of developing high fasting glucose and high triglyceride levels. Consuming mixed types of alcohol increased the risk of developing MS and abdominal obesity. Meanwhile, those who drank liquor or wine had a greater risk of developing high triglyceride or high fasting glucose levels, respectively. In conclusion, alcohol consumption dose-dependently increased the risk of developing MS and some of its individual components while dose-dependently decreasing the risk of developing low HDL-C levels. The type of alcoholic beverage had different effects on the development of the individual components of MS.

© 2012 Elsevier Inc. All rights reserved.

Keywords: Metabolic syndrome; Alcohol; Ethanol; Triglyceride; Obesity; Glucose Abbreviations: HDL-C, high-density lipoprotein cholesterol; MR, metabolic syndrome.

^{*} Corresponding author. Department of Family Medicine, China Medical University Hospital, Taichung 40447, Taiwan. Tel.: +886 4 22062121x7629; fax: +886 4 22335695.

1. Introduction

Metabolic syndrome is a cluster of diseases characterized by abdominal obesity, hypertriglyceridemia, low levels of high-density lipoprotein cholesterol (HDL-C), elevated blood glucose levels, and high blood pressure. Previous reports have shown that metabolic syndrome is associated with increased all-cause mortality [1] and the development of type 2 diabetes mellitus [2]. It is believed that having a sedentary lifestyle is a key determinant in the occurrence of metabolic syndrome. Other modifiable risk factors such as diet and cigarette smoking [3] also play an important role in the development of metabolic syndrome in individuals with a genetic predisposition for this group of diseases.

Alcohol has both adverse and protective effects on the individual components of metabolic syndrome. Previous reports have shown that alcohol consumption is positively associated with having abdominal obesity [4-6], high triglyceride levels [7,8], and high blood pressure [8-10]. However, quite a few studies have demonstrated that alcohol consumption has a protective effect on the development of metabolic syndrome by increasing HDL-C levels [7,8,11,12]. Although the results from some studies have shown a U-shaped association between alcohol consumption and plasma glucose levels, that relationship was based on a small number of studies that had different study designs and definitions [13,14]. Taking the inconsistent results of previous studies into consideration, it is unclear whether alcohol consumption contributes to the development of metabolic syndrome. To make a cogent recommendation about alcohol consumption to patients with cardiometabolic diseases, it is necessary to clarify the association of alcohol consumption with metabolic syndrome and its individual components. Because alcohol increases some risk factors for metabolic syndrome, we hypothesize that alcohol consumption increases the risk of developing metabolic syndrome and that different types of alcoholic beverages have different effects on the development of metabolic syndrome and its individual components. The aim of this study was to investigate the relationship among alcohol consumption, metabolic syndrome, and its individual components.

2. Methods and materials

2.1. Participants

The study subjects composed of 2 different populations. The first population was from our previous community-based, cross-sectional study, conducted from October 2004 to September 2005, which estimated the prevalence of metabolic syndrome in Taichung city [15]. In that study, based on individuals' records from the Bureau of Households in Taichung city, we used a 2-stage sampling design to choose residents and ensured that the sampling rate was proportional to the number of residents within each stage. Among the 3530 eligible subjects, 2359 subjects (1147 men

and 1212 women) agreed to participate, giving us a response rate of 66.83%. Using the same questionnaire, we recruited the second population (1256 men and 1231 women) during routine physical examinations at the Department of Family Medicine at China Medical University Hospital from January 2006 to December 2006. In this study, we restricted our analyses to men because the proportion of Taiwanese women who drank alcohol was too small. The final analysis was conducted with 2358 men after excluding 45 subjects with incomplete data for any of the following variables: alcohol drinking status, smoking status, physical activity, and daily energy intake or the parameters for the diagnosis of metabolic syndrome. Informed consent was obtained from each participant, and the study was approved by the Human Research Committee of the China Medical University Hospital.

2.2. Questionnaire

The data were collected from self-reported nutrition and lifestyle questionnaires. Items in the questionnaire explored basic demographic data, previous and current disease status, family history of disease, smoking habits, alcohol consumption, betel nut chewing, and physical activity status. The nutrition survey used a food intake frequency method to assess daily energy and nutrition intakes. Alcohol consumption was assessed by the type of beverages consumed, the age at onset of drinking (or the age when the subject began to abstain), drinking frequency, and the average amount of alcohol per drink. Individuals who drank at least once per week for 6 consecutive months were classified as current drinkers. Physical activity was measured by the frequency, duration, and intensity of walking, jogging, running, bicycle riding, swimming, aerobics, aerobic dancing, and other types of dancing, as well as the frequency and duration of time the individual spent playing tennis, table tennis, golf, basketball, or badminton.

2.3. Anthropometric measurements and laboratory analyses

All participating subjects reported to the outpatient clinic of the Department of Family Medicine after an overnight fast. They were weighed in light clothing, and their heights were measured. Waist circumference was measured in a horizontal plane midway between the inferior margin of the last rib and the crest of the ileum. The circumference was measured to the nearest 1 mm. Blood pressure was recorded from the right arm after the participant sat at rest for a period of 20 minutes. The mean of 2 blood pressure recordings was used for statistical analyses. Fasting blood samples were drawn between 8:00 and 10:00 AM.

The plasma glucose level was determined using a glucose oxidase method (Astra-8; Beckman, Fullerton, Calif, USA). Plasma lipids were determined using an enzymatic colorimetric method (Synchron LX-20; Beckman Coulter, Brea, Calif, USA).

2.4. Diagnosis of metabolic syndrome

Metabolic syndrome was diagnosed using the American Heart Association/National Heart, Lung, and Blood Institute's criteria with minor modifications [16]: serum triglyceride level, ≥1.69 mmol/L (150 mg/dL) or currently taking hypolipidemic agents; serum HDL-C level, ≥1.03 mmol/L (40 mg/dL); blood pressure, ≥130/85 mm Hg or currently taking antihypertensive medication; fasting plasma glucose, ≥5.6 mmol/L (100 mg/dL) or currently taking oral antidiabetic medication; and waist circumference, ≥90 cm.

2.5. Statistical analyses

All data are presented as the means \pm SD, except alcohol drinking status, which was separated by the median (lower quantile–upper quantile). Using drinking status as a factor, continuous variables were analyzed with Student t test, and nominal variables were analyzed with the χ^2 test to determine whether there were significant differences between the groups. A multiple logistic regression analysis was used to calculate the odds ratios, and the linear trend was evaluated using the trend test. Subjects without data for their education level (n = 164) or household income (n = 361) were included in the multiple logistic regression analysis and the trend test. A P value of less than .05 represented a statistically significant difference between the compared data sets. All analyses were performed with the statistical package SAS for Windows (version 8.1; SAS Institute, Cary, NC, USA).

3. Results

The characteristics of the study subjects are given in Table 1. Among the 2358 men, 1430 subjects (61%) had never drunk alcohol, and 928 subjects (39%) were current drinkers. The current drinkers were younger than the never drinkers. The percentage of subjects with an education level higher than 12th grade was higher among the never drinkers than among the current drinkers, although the household income was similar between the 2 groups. The average daily amount of alcohol drinking in the current drinkers was 35.71 g. The drinking amount was the highest among liquor drinkers. The percentage of subjects who currently smoke was higher among the current drinkers. These subjects also had higher levels of physical activity and fat intake than did the never drinkers. Fiber intake, however, was lower among the current drinkers. There were significant differences between the 2 groups in body mass index and waist circumference. The current drinkers had a higher proportion of subjects with high triglyceride levels than the never drinkers, but the percentage of subjects with low HDL-C levels was similar between the 2 groups. The proportion of subjects with high blood pressure and high fasting plasma glucose was also similar between the never drinkers and the current drinkers. Metabolic syndrome was more prevalent in the current drinkers than in the never drinkers.

Table 1 Characteristics of the study subjects categorized by alcohol consumption

Characteristics (n = 2358)	Never drinkers	Current drinkers	P
	(n = 1430)	(n = 928)	
Age (y)	51.95 ± 12.78	50.47 ± 10.19	.0018
Education level (grade) ^a			<.0001
<9	236 (17.81)	178 (20.48)	
9-12	551 (41.58)	457 (52.59)	
>12	538 (40.60)	234 (26.93)	
Household income (USD/mo) ^b			.3801
<1250	269 (22.47)	159 (19.88)	
1250-4000	562 (46.95)	390 (48.75)	
>4000	366 (30.58)	251 (31.38)	
Alcohol drinking status ^c	(,	(
Total amount (g/d)	_	35.71	
rotal amount (g. a)		(14.29-107.14)	
Beer drinker (g/d)	_	35.71	
Beer drinker (g/d)		(12.50-107.14)	
Wine drinker (g/d)	_	26.79	
wille drilliker (g/d)		(10.71-71.43)	
Liquor drinker (g/d)		42.86	
Liquoi diffikei (g/d)	_		
Missad duintran (a/d)		(17.86-107.14)	
Mixed drinker (g/d)	_	28.57	
C1-i		(13.39-89.29)	< 0001
Smoking status	977 ((1.27)	200 (21.25)	<.0001
Never	876 (61.26)	290 (31.25)	
Former smoker	180 (12.59)	171 (18.43)	
Current smoker	374 (26.15)	467 (50.32)	0006
Physical activity (MET-h/wk)	10.97 ± 18.02	14.02 ± 22.65	.0006
Daily energy intake			
Total calories (kJ)	10.62 ± 2.43	10.56 ± 2.40	.5692
Carbohydrate (g)	476.80 ± 111.71	469.22 ± 110.16	.1055
Fat (g)	31.84 ± 10.79	33.04 ± 11.61	.0116
Protein (g)	78.72 ± 19.77	80.24 ± 20.34	.0715
Fiber (g)	6.58 ± 2.05	6.41 ± 2.06	.0483
Anthropometric measures			
Body mass index (kg/m ²)	24.23 ± 3.25	24.78 ± 3.18	<.0001
Total cholesterol (mmol/L)	5.13 ± 0.95	5.25 ± 0.96	.0017
Metabolic syndrome			
parameters			
Waist circumference ≧90 cm	375 (26.22)	298 (32.11)	.0021
Triglycerides ≥1.69 mmol/L	437 (30.56)	361 (38.90)	<.0001
(150 mg/dL) or			
on medication			
HDL-C <1.03 mmol/L	859 (60.07)	528 (56.90)	.1339
(40 mg/dL)			
Blood pressure	572 (40.00)	385 (41.49)	.4923
≥130/85 mm Hg			
or on medication			
Fasting plasma glucose	412 (28.81)	281 (30.28)	.4592
≥5.6 mmol/L (100 mg/dL)	, ,	, ,	
or on medication			
Prevalence of metabolic	447 (31.26)	333 (35.88)	.0223
syndrome	. /	, ,	

Data are presented as means \pm SD or n (%). MET indicates metabolic equivalent.

 $^{^{\}rm a}$ One hundred sixty-four subjects (n = 105 in never group; n = 59 in current group) without data for education level.

^b Three hundred sixty-one subjects (n = 233 in never group; n = 128 in current group) without data for household income.

^c The alcohol drinking amount was separated at the median (lower-upper quantile).

Table 2 Association of metabolic syndrome and its components with current alcohol drinking

	Never	Current $(n = 928)$		
	(n = 1430)	Crude OR	Adjusted OR	
Metabolic syndrome	1.00	1.23 (1.03-1.47)	1.24 (1.02-1.50)	
Abdominal obesity	1.00	1.33 (1.11-1.60)	1.27 (1.05-1.55)	
High triglyceride	1.00	1.45 (1.22-1.72)	1.29 (1.07-1.57)	
Low HDL-C	1.00	0.88 (0.74-1.04)	0.80 (0.67-0.96)	
High fasting glucose	1.00	1.07 (0.90-1.29)	1.15 (0.94-1.41)	
High blood pressure	1.00	1.06 (0.90-1.26)	1.19 (0.98-1.43)	

Presented with odds ratios (ORs) (95% confidence interval). The model is adjusted for age, education level, smoking status, physical activity, fat intake, fiber intake, and lipid medication. Subjects without data for education level or household income were included in the analyses. A multiple logistic regression analysis was used to calculate the OR. The current drinkers were at a significantly higher risk of developing metabolic syndrome, abdominal obesity, and high triglyceride levels, but they were at a lower risk of developing low HDL-C levels than the never drinkers. The risk of developing high fasting glucose levels and high blood pressure was similar between the 2 groups.

After controlling for other covariates, the current drinkers were at a significantly higher risk of developing metabolic syndrome (P = .0307), abdominal obesity (P = .0154), and high triglyceride levels (P = .0090), but they were at a lower risk of developing low HDL-C levels (P = .0175) than the never drinkers. The risk of developing high fasting glucose levels and high blood pressure was similar between the 2 groups (Table 2).

There was a significant, dose-dependent relationship among the amount of alcohol consumed, the development of metabolic syndrome (P=.0312 for trend), high triglyceride levels (P=.0004 for trend), and high fasting glucose levels (P=.0058 for trend). The dose-dependent relationship between the amount of alcohol consumed and low HDL-C levels was reversed (P<.0001 for trend) (Table 3). The dose associated with the development of

low HDL-C levels was ≥ 50 g/d (P < .0001); however, that dose of alcohol increased the risk of developing high fasting glucose levels (P = .0090) and high triglyceride levels (P = .0055) (Table 3).

As shown in Table 4, having a mixed type of alcohol consumption was associated with the development of metabolic syndrome (P = .0397) and abdominal obesity (P = .0069). Liquor drinking significantly increased the likelihood of developing high triglyceride levels (P = .0003), and wine consumption was associated with a greater risk of developing high fasting glucose levels (P = .0408).

4. Discussion

This study showed that alcohol consumption increased the risk of developing metabolic syndrome and some of its individual components in a dose-dependent manner. Triglyceride levels were significantly higher in subjects who consumed ≥10 g of alcohol per day. Drinking more than 50 g of alcohol per day significantly decreased the risk of developing low HDL-C levels but increased the risk of developing high fasting glucose levels. The type of alcoholic beverages consumed was not related to the development of metabolic syndrome. Consuming mixed types of alcohol, however, increased the risk of developing metabolic syndrome.

The results from previous studies on the relationship between alcohol consumption and the development of metabolic syndrome are inconsistent. Some studies have reported that the association is positively linear [17,18], others have demonstrated that the relationship is inversely linear [19,20], some have seen a J-shaped relationship [21], and 1 study showed that there was no relationship between alcohol consumption and metabolic syndrome [22]. In this study, we demonstrated a positive, linear relationship between alcohol consumption and metabolic syndrome. The discrepancies in past study results may be partly attributed to different study

Table 3
Association of metabolic syndrome and its components with an alcohol drinking amount

Drinking amount (g/d)	Never	Current drinkers (n = 928)				P for
		> 0, <10	≧10, <30	≧30, <50	≥ 50	trend
n	1430	491	231	77	129	
Metabolic syndrome	1.00	1.18 (0.94-1.49)	1.25 (0.92-1.71)	1.53 (0.94-2.50)	1.32 (0.87-1.95)	.0312
Abdominal obesity	1.00	1.34 (1.06-1.69)	1.08 (0.78-1.49)	1.50 (0.91-2.47)	1.26 (0.84-1.90)	.0806
High triglycerides	1.00	1.10 (0.87-1.39)	1.48 (1.09-2.02)	1.57 (0.96-2.55)	1.74 (1.18-2.56)	.0004
Low HDL-C	1.00	0.95 (0.77-1.18)	0.78 (0.58-1.05)	0.79 (0.49-1.27)	0.41 (0.28-0.60)	<.0001
High fasting glucose	1.00	1.00 (0.78-1.27)	1.23 (0.89-1.69)	1.42 (0.85-2.37)	1.72 (1.15-2.59)	.0058
High blood pressure	1.00	1.18 (0.94-1.48)	1.24 (0.91-1.67)	1.07 (0.65-1.76)	1.19 (0.80-1.77)	.1845

Presented with adjusted OR (95% confidence interval). The model is adjusted for age, education level, smoking status, physical activity, fat intake, fiber intake, and lipid medication. Subjects without data for education level or household income were included in the analyses. A multiple logistic regression analysis was used to calculate the OR, and the linear trend was evaluated using the trend test. There was a significant, dose-dependent relationship among the amount of alcohol consumed and the development of metabolic syndrome, high triglyceride levels, and high fasting glucose levels; whereas low HDL-C levels had the reversed relationship. The dose associated with developing low HDL-C levels was ≥ 50 g/d; however, that dose increased the risk of developing high fasting glucose levels and high triglyceride levels.

Table 4
Association of metabolic syndrome and its components with alcoholic beverages

	Adjusted OR (95% CI)				
	Never	Beer	Wine	Liquor	Mixed types
n	1430	106	160	466	196
Metabolic syndrome	1	0.90 (0.57-1.43)	1.33 (0.93-1.89)	1.17 (0.91-1.50)	1.41 (1.02-1.96)
Abdominal obesity	1	1.21 (0.77-1.89)	1.18 (0.82-1.70)	1.20 (0.93-1.55)	1.58 (1.13-2.20)
High triglyceride	1	0.85 (0.53-1.35)	1.19 (0.82-1.72)	1.58 (1.23-2.02)	1.21 (0.86-1.69)
Low HDL-C	1	0.78 (0.52-1.18)	0.93 (0.66-1.30)	0.86 (0.68-1.10)	1.01 (0.73-1.39)
High fasting glucose	1	1.04 (0.65-1.67)	1.45 (1.02-2.07)	0.98 (0.75-1.28)	1.26 (0.89-1.77)
High blood pressure	1	0.93 (0.60-1.44)	1.15 (0.81-1.64)	1.11 (0.87-1.42)	1.13 (0.81-1.57)

A multiple logistic regression analysis was used to calculate the OR. The model is adjusted for age, education level, smoking status, physical activity, fat intake, fiber intake, drinking amount, and lipid medication. Subjects without data for education level or household income were included in the analyses. Consuming mixed types of alcohol was associated with the development of metabolic syndrome and abdominal obesity. Meanwhile, those that consumed liquor or wine had a greater risk of developing high triglyceride or high fasting glucose levels, respectively. OR indicates odds ratio; CI, confidence interval.

populations and different consumption patterns. Ethnic differences may also play a role in the discrepancy. For example, a previous report showed that an individual's HDL₂-C level was positively associated with alcohol consumption in white Americans but not in African Americans [23]. That study also demonstrated that heavy alcohol consumption was associated with higher triglyceride levels in African Americans but not in white Americans [23].

The results of this study were consistent with previous reports that alcohol consumption increases the risk of developing abdominal obesity [4-6] and high triglyceride levels [7,8] while lowering the risk of having low HDL-C levels [7,8,11,12]; however, our results contrast with those of some studies that showed that current drinkers had a lower risk of developing abdominal obesity [24,25] and high blood pressure [8-10]. A meta-analysis showed that moderate alcohol consumption lowers the risk of developing type 2 diabetes but that this effect disappears in subjects who drank ≥48 g of alcohol per day [9]. Similarly, we found that the consumption of more than 50 g of alcohol per day significantly increased the subject's risk of developing high fasting glucose levels.

Freiberg et al [19] showed that the risk of developing metabolic syndrome differed depending on the type of alcoholic beverages consumed. In contrast, Djousse et al [20] demonstrated that alcohol consumption was associated with a lower prevalence of metabolic syndrome irrespective of the type of alcoholic beverages that were consumed. In this study, the association between alcohol consumption and metabolic syndrome was not related to type of alcoholic beverages consumed. The reason for the discrepancy among these studies is not clear.

This study had some limitations. First, the smoking and alcohol drinking statuses were based on the results of self-reported questionnaires; therefore, some of the individuals may have been misclassified. Second, this was a cross-sectional study, and we did not evaluate or consider longitudinal changes in the participants' habits. Third, although we adjusted for a variety of potential confounders, residual confounding factors are still possible.

In conclusion, the results of this study indicate that the risk of developing metabolic syndrome is greater among current drinkers than among never drinkers. In addition, the increased risk of developing metabolic syndrome and many of its individual components, namely, high triglyceride and fasting glucose levels, was dose dependent. The type of alcoholic beverages consumed had different effects on the development of the individual components of metabolic syndrome; however, it was not related to the development of metabolic syndrome. The fact that the consumption of a moderate amount of alcoholic beverages has been shown to have protective cardiovascular effects may outweigh the negative effects of consuming alcohol.

Acknowledgment

This work was supported by a grant from the China Medical University Hospital (DMR-98-024) in Taichung, Taiwan. The authors declare no conflicts of interest.

References

- Lakka HM, Laaksonen DE, Lakka TA, et al. The metabolic syndrome and total and cardiovascular disease mortality in middle-aged men. JAMA 2002;288:2709-16.
- [2] Hanson RL, Imperatore G, Bennett PH, Knowler WC. Components of the "metabolic syndrome" and the incidence of type 2 diabetes. Diabetes 2002;51:3120-7.
- [3] Chen CC, Li TC, Chang PC, et al. Association among cigarette smoking, metabolic syndrome, and individual components: the metabolic syndrome study in Taiwan. Metabolism 2008;57:544-58.
- [4] Tolstrup JS, Heitmann BL, Tjonneland AM, Overvad OK, Sorensen TI, Gronbaek MN. The relation between drinking pattern and body mass index and waist and hip circumference. Int J Obes (Lond) 2005; 29:490-7.
- [5] Schroder H, Morales-Molina JA, Bermejo S, et al. Relationship of abdominal obesity with alcohol consumption at population scale. Eur J Nutr 2007;46:369-76.
- [6] Riserus U, Ingelsson E. Alcohol intake, insulin resistance, and abdominal obesity in elderly men. Obesity 2007;15:1766-73.
- [7] Ruixing Y, Shangling P, Hong C, et al. Diet, alcohol consumption, and serum lipid levels of the middle-aged and elderly in the Guangxi Bai Ku Yao and Han population. Alcohol 2008;42:219-29.

- [8] Lee KS, Park CY, Meng KH, et al. The association of cigarette smoking and alcohol consumption with other cardiovascular risk factors in men from Seoul, Korea. Ann Epidemiol 1998;8: 31-8
- [9] Taylor B, Irving HM, Baliunas D, et al. Alcohol and hypertension: gender difference in dose-response relationships determined through systematic and meta-analysis. Addiction 2009;104:1981-90.
- [10] Sesso HD, Cook NR, Buring JE, Manson JE, Gaziano JM. Alcohol consumption and the risk of hypertension in women and men. Hypertension 2008;51:1080-7.
- [11] Linn S, Carroll M, Johnson C, Fulwood R, Kalsbeek W, Briefel R. High-density lipoprotein cholesterol and alcohol consumption in US white and black adults: data from NHANES II. Am J Public Health 1993;83:811-6.
- [12] Suh I, Shaten BJ, Cutler JA, Kuller LH. Alcohol use and mortality from coronary heart disease: the role of high-density lipoprotein cholesterol. Ann Int Med 1992;116:881-7.
- [13] Koppes LL, Dekker JM, Hendriks HF, Bouter LM, Heine RJ. Moderate alcohol consumption lowers the risk of type 2 diabetes: a meta-analysis of prospective observational studies. Diabetes Care 2005;28:719-25.
- [14] Carlsson S, Hammar N, Grill V. Alcohol consumption and type 2 diabetes: meta-analysis of epidemiological studies indicates a U-shaped relationship. Diabetologia 2005;48:1051-4.
- [15] Lin CC, Liu CS, Lai MM, et al. Metabolic syndrome in a Taiwanese metropolitan adult population. BMC Public Health 2007;7:239-43.
- [16] Grundy SM, Cleeman JI, Daniels SR, et al. Diagnosis and management of the metabolic syndrome: an American Heart Association/National Heart, Lung, and Blood Institute scientific statement: executive summary. Circulation 2005;112:285-90.

- [17] Fan AZ, Russell M, Dorn J, et al. Lifetime alcohol drinking pattern is related to the prevalence of metabolic syndrome. The Western New York Health Study (WNYHS). Eur J Epidemiol 2006;21:129-38.
- [18] Baik I, Shin C. Prospective study of alcohol consumption and metabolic syndrome. Am J Clin Nutr 2008;87:1455-63.
- [19] Freiberg MS, Cabral HJ, Heeren TC, Vasan RS, Curtis Ellison R. Alcohol consumption and the prevalence of the metabolic syndrome in the US: a cross-sectional analysis of data from the Third National Health and Nutrition Examination Survey. Diabetes Care 2004;27: 2954-9.
- [20] Djousse L, Arnett DK, Eckfeldt JH, Province MA, Singer MR, Ellison RC. Alcohol consumption and metabolic syndrome: does the type of beverage matter? Obes Res 2004;12:1375-85.
- [21] 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 2004;80:217-24.
- [22] Zhu S, St-Onge MP, Heshka S, Heymsfield SB. Lifestyle behaviors associated with lower risk of having the metabolic syndrome. Metabolism 2004;53:1503-11.
- [23] Volcik KA, Ballantyne CM, Fuchs FD, Sharrett AR, Boerwinkle E. Relationship of alcohol consumption and type of alcoholic beverage consumed with plasma lipid levels: differences between whites and African Americans of the ARIC study. Ann Epidemiol 2008;18:101-7.
- [24] Rohrer JE, Rohland BM, Deison A, Way A. Frequency of alcohol use and obesity in community medicine patients. BMC Family Practice 2005:6:17-25.
- [25] Arif AA, Rohrer JE. Patterns of alcohol drinking and its association with obesity: data from the third national health and nutrition examination survey, 1988-1994. BMC Public Health 2005;5:126-31.