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Relationship Between Alcohol Consumption and Serum Lipid Profiles Among Middle-Aged Population in China: A Multiple-Center Cardiovascular Epidemiological Study

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Abstract

We assessed the relationship between alcohol consumption and serum lipids in a middle-aged Chinese population. The overall prevalence of drinking among 10 154 participants was 34.07% in males and 3.61% in females. Heavy alcohol drinkers ($\geq$ 30 g/d) tended to be older, smokers, hypertensive, do heavy physical activity, and have a lower body mass index. Levels of high-density lipoprotein cholesterol (HDL-C), apolipoprotein (apo) A1, low-density lipoprotein cholesterol–HDL-C ratio, and apo B–apo A1 ratio rose with increase in alcohol intake in males. An increase of 0.27 mmol/L in triglycerides and a decrease of 2.10 mg/dL in lipoprotein(a), Lp(a), were observed in male alcohol drinkers who consumed $\geq$30 g alcohol/d compared with abstainers after controlling for all confounders. Levels of total cholesterol, HDL-C, and apo A1 increased with increase in alcohol intake in both genders and Lp(a) decreased with the increase in alcohol intake in males.

Keywords

alcohol, serum lipid, association, China

Introduction

Dyslipidemia is a cardiovascular (CV) risk factor, and CV disease is a major cause of morbidity and mortality worldwide, even in developing countries like China.[1,2] There is evidence of an increase in the use of alcohol and consequent alcohol-related health issues.[3,4] Heavy drinking increases CV risk, but whether light to moderate alcohol consumption has a beneficial effect on CV risk remains uncertain.[5-8]

The effect of alcohol intake on the risk of CV disease may through changes in the lipids level.[9,10] Previous studies have shown that high total cholesterol (TC), high triglyceride (TG), low high-density lipoprotein cholesterol (HDL-C), and high low-density lipoprotein cholesterol (LDL-C) are risk factors for CV disease.[11,12] Several lipid-related indices have been proposed, such as the LDL-C/HDL-C and TG/HDL-C ratios, as predictors of CV risk.[13-15] However, studies that focused on the association between alcohol consumption and lipid-related indices in Chinese populations are sparse. On the other hand, given that CV disease has become an important health problem in China[16] and the current drinking rate of people in China was high (39.6% for males and 4.5% for females), it is important to investigate whether alcohol consumption affects dyslipidemia. Therefore, we investigated the association between alcohol consumption and serum lipids.

Participants and Methods

Participants

Data were derived from a cross-sectional survey on risk factors for CV disease, which was conducted in 2009 to 2010.[17] There were 12 different research populations, including southern and northern and urban and rural in different parts of China, selected based on economic and social development level and the basis of previous research. About 1000 participants, aged

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35 to 64, half male and half female, were recruited in each population using cluster random sampling method. A total of 14 046 inhabitants were invited to our survey, and of them, 11 623 participated in our survey; the response rate was 82.75%. Participants who reported taking cholesterol-lowering drugs or had insufficient data were excluded. There were 10 154 (85.94%) participants eligible for analysis. Informed consent was obtained and approval for the survey granted by the Fuwai hospital Ethics Review Board.

Data Collection

The survey was performed according to unification protocol designed by coordination center. Demographic and sociocultural data, biochemical data, medical history, and physical examination data were collected by trained research staff using international standardized examination and measurement. Overnight fasting blood specimens were collected by venipuncture for measurement of serum lipids, plasma glucose (GLU), and so on.

Height, Weight, and Body Mass Index

Weight and height were measured twice during the interview. Height without shoes was measured using a standard right angle device, and weight was measured by using Omron HBF-306C Body Fat Analyzer (Kyoto, Japan). Body mass index (BMI) was defined as weight divided by the square of height (kg/m^2).

Blood Pressure

Blood pressure (BP) was measured 3 times with participants in a seated position, after at least 5 minutes rest, using a calibrated mercury sphygmomanometer. Cuff sizes were chosen based on arm circumference. Participants were advised to avoid cigarette, smoking, alcohol, caffeinated beverages, and exercise for at least 30 minutes before measurement.

Blood Specimen

Overnight fasting blood specimens were collected by venipuncture for measurement of serum lipids and plasma GLU. Blood specimens were centrifuged and plasma was stored at $-80^{\circ}C$ until laboratory assay. Plasma GLU was measured by the glucose oxidase-peroxidase method; concentrations of TC were measured by glucose oxidase-polymerization method; TG was assessed by method of glycerol-3-phosphate oxidase-phenol + aminophenazone; HDL-C and LDL-C were measured by synthetic polymer/detergent method; and apo A1and apo B were measured by turbidimetric immunoassay method. Lipoprotein(a), Lp(a), was measured by liposome immunoassay method. All biochemical indicators were analyzed by Hitachi HITACH17080 (Hitachi, Ltd., Tokyo, Japan) automatic biochemistry analyzer at the China Isotope & Radiation Corporation/Beijing CIC clinical laboratory, Beijing, China.

Table 1. Basic Characteristics of Participants.

Characteristics	Male (n = 4700)	Female (n = 5454)
Age, years	50.0 ± 8.2	49.9 ± 7.9
Body mass index, kg/m^2	24.4 ± 3.5	24.7 ± 3.7
Smoking, n (%)	3005 (63.9)	389 (7.1)
Education of high school and above, n (%)	2798 (59.5)	2680 (49.1)
Physical activity, n (%)		
Light	2498 (53.1)	4065 (74.5)
Moderate	1334 (28.4)	894 (16.4)
Heavy	868 (18.5)	495 (9.1)
Hypertension, n (%)	1956 (41.7)	1935 (35.5)
Diabetes, n (%)	484 (10.3)	461 (8.4)
Alcohol consumption categories, n (%)		
Never	3082 (65.9)	5257 (96.4)
0.1-9.9	415 (8.8)	127 (2.3)
10-19.9	301 (6.4)	31 (0.6)
20-29.9	201 (4.3)	10 (0.2)
≥30	701 (14.9)	29 (0.5)

Definitions

Diabetes was defined as a fasting plasma GLU level ≥7.0 mmol/L or self-reported treatment with antidiabetic medication (insulin or oral hypoglycemic agents). Hypertension was defined as an average (from 3 measurements) systolic BP ≥140 mm Hg or an average diastolic BP ≥90 mm Hg or self-reported treatment with antihypertensive medication. Smoking was defined as use of at least 20 packets of cigarettes or 0.5 kg of leaf tobacco in their lifetime or smoking 1 cigarette/d at least for 1 year. The amount of tobacco smoked was classified as cigarettes or cigarette equivalent/d (about 1 g of tobacco per commercial cigarette). Patients were asked about their average frequency and amount of alcoholic beverage intake, which were converted into the amount of pure alcohol consumed per day.

Statistical Analysis

Participants were categorized into 5 groups according to average alcohol consumption/d: 0, 0.1-9.9, 10.0-19.9, 20.0-29.9, and ≥30 g/d groups. We calculated the mean or percentage of basic participant characteristics by alcohol consumption categories. Linear regression models were used to estimate the association between alcohol intake and blood lipid level. The blood lipid levels in each category of total alcohol intake were compared with lipid levels in never drinkers (reference group). Age was adjusted in model 1. In the second model, BMI, education, cigarette smoking status, physical activity, hypertension, and diabetes were added. All statistical analyses were performed using SAS 9.2 (SAS Institute Inc, Cary, North Carolina). A 2-sided $P < .05$ was considered significant.

Results

Overall, a greater proportion (34.07%) of males reported consuming alcohol ≥1 g/d compared to females (3.61%). Basic

Table 2. Basic Characteristics of Male Participants According to Alcohol Intake Categories.

Characteristics	Never (n = 3082)	0.1-9.9 (n = 415)	10-19.9 (n = 301)	20-29.9 (n = 201)	≥30 (n = 701)	*P* for Trend
Age, years	50.1 ± 8.3	48.1 ± 8.3	48.4 ± 8.1	49.1 ± 7.7	51.4 ± 7.5	<.001
Body mass index, kg/m^2	24.5 ± 3.6	24.2 ± 3.3	24.3 ± 3.6	24.3 ± 3.7	24.2 ± 3.3	<.001
Smoking, n (%)	1766 (57.3)	302 (32.8)	233 (77.4)	146 (72.5)	558 (76.6)	<.001
Education of high school and above, n (%)	1842 (59.8)	289 (69.6)	181 (60.1)	129 (64.2)	357 (50.9)	<.001
Physical activity, n (%)						
Light	1707 (55.4)	229 (55.2)	147 (48.8)	98 (48.8)	317 (45.2)	<.001
Moderate	809 (26.2)	124 (29.9)	101 (33.5)	80 (39.8)	220 (31.4)	
Heavy	566 (18.4)	62 (14.9)	53 (17.6)	23 (11.4)	164 (23.4)	
Hypertension, n (%)	1245 (40.4)	152 (36.6)	131 (43.5)	94 (46.8)	336 (47.9)	<.001
Diabetes, n (%)	330 (10.7)	36 (8.7)	27 (9.0)	12 (6.0)	79 (11.3)	.663

Table 3. Blood Lipids Level According to Alcohol Consumption Categories Among Male Participants.

Characteristics	Never	0.1-9.9	10-19.9	20-29.9	≥30	*P*
TC, mmol/L	4.77 ± 0.91	4.84 ± 0.88	4.86 ± 0.92	4.86 ± 0.93	5.01 ± 0.95	<.001
TG, mmol/L	1.62 ± 1.15	1.82 ± 1.36	1.78 ± 1.18	1.84 ± 1.31	1.76 ± 1.38	<.001
LDL-C, mmol/L	2.80 ± 0.78	2.74 ± 0.77	2.72 ± 0.81	2.71 ± 0.77	2.79 ± 0.81	.166
HDL-C, mmol/L	1.27 ± 0.27	1.33 ± 0.28	1.36 ± 0.31	1.36 ± 0.32	1.47 ± 0.36	<.001
Apo A1, mg/dL	132 ± 24	140 ± 24	144 ± 26	144 ± 27	156 ± 35	<.001
Apo B, mg/dL	93 ± 21	93 ± 21	93 ± 22	92 ± 21	95 ± 21	459
Lp(a), mg/dL	14.00 ± 15.50	13.25 ± 15.14	13.94 ± 15.19	12.27 ± 15.12	12.22 ± 14.44	.044
Non–HDL-C, mmol/L	3.50 ± 0.88	3.51 ± 0.87	3.50 ± 0.90	3.49 ± 0.90	3.55 ± 0.94	.856
LDL-C/HDL-C	2.29 ± 0.74	2.13 ± 0.70	2.08 ± 0.73	2.07 ± 0.66	2.02 ± 0.76	<.001
TG/HDL-C	1.40 ± 1.24	1.49 ± 1.32	1.42 ± 1.10	1.50 ± 1.26	1.33 ± 1.22	.225
Apo B/apo A1	0.73 ± 0.20	0.68 ± 0.19	0.67 ± 0.20	0.66 ± 0.19	0.64 ± 0.21	<.001

Abbreviations: TG, total cholesterol; TC, triglyceride; LDL-C, low-density lipoprotein cholesterol; HDL-C, high-density lipoprotein cholesterol; Apo A1, apolipoprotein A1; Apo B, apolipoprotein B; Lp(a), lipoprotein(a); SD, standard deviation; N, number.

characteristics according to categories of alcohol consumption among 5454 female and 4077 male are presented in Table 1. Heavy alcohol drinkers (≥30 g/d) tended to be older, smokers, hypertensive, do heavy physical activity, and have a lower BMI in males (*P* for trend <.001: Table 2). Additionally, males with high school education and more tend to consume light to moderate alcohol (1-30 g/d). Due to the lower proportion of drinkers (3.61%) in the female population, the association between alcohol consumption and lipid profiles was analyzed only in males.

Univariate analysis showed the mean lipid levels associated with alcohol consumption (Table 3). Total cholesterol, TG, HDL-C, and apo A1 levels rose with increase in alcohol intake, and the Lp(a), LDL-C–HDL-C ratio, and apo B–apo A1 ratio decreased with increase in the alcohol intake in males (all *P* for trend <.05).

Table 4 presents multiple regression coefficients for total alcohol intake and blood lipids. For TC, an increase of 0.27 mmol/L was observed in male alcohol drinkers who consumed ≥30 g alcohol/d compared with abstainers after controlling for all confounders. Alcohol intake was positively associated with TG, HDL-C, and apo A1, and an inverse association was observed between total alcohol intake,LDL-C/HDL-C and apo B/apo A1 in males after adjustment for age and other confounders. For Lp(a), a decrease of 2.10 mg/dL was observed in male alcohol drinkers who consumed ≥30 g alcohol/d compared with abstainers after controlling for confounders.

Discussion

Accumulating evidence indicates that light to moderate drinking may significantly reduce the risk of coronary heart disease (CHD) and all-cause mortality; this link is probably J-shaped.[18,19] A recent meta-analysis found that the association between alcohol intake and stroke morbidity and mortality is J-shaped, namely, low alcohol intake is associated with a reduced risk of stroke morbidity and mortality, whereas heavy alcohol intake is associated with an increased risk of total stroke.[20] In previous studies evaluating whether different alcoholic beverages would protect against CV disease, a J-shaped relationship for increasing wine consumption and vascular risk was found; however, this association was controversial for beer or spirits.[21] In our study, most of the individuals were drinking liquor (68.2% drinking liquor, 31.8% drinking beer, red wine, etc).

The associations between alcohol consumption and the risk of CV disease, including myocardial infarction and CHD, are mediated, at least in part, by the differential influence on the levels of HDL-C and non-HDL-C.[22,23] Our data from middle-aged

Table 4. Regression Coefficients for the Association of Lipid Concentration and Alcohol Consumption Categories Among Male Participants (g/d).[a,b]

Characteristics	Never	0.1-9.9 g/d	10-19.9 g/d	20-29.9 g/d	≥30 g/d	*P* for Trend
TC, mmol/L						
Model 1	Reference	0.071 (0.139)	0.097 (0.080)	0.088 (0.187)	0.239 (<.0001)	<.001
Model 2	Reference	0.068 (0.146)	0.103 (0.057)	0.089 (0.175)	0.269 (<.0001)	<.001
TG, mmol/L						
Model 1	Reference	0.174 (0.006)	0.138 (0.059)	0.207 (0.019)	0.158 (0.002)	<.001
Model 2	Reference	0.183 (0.003)	0.132 (0.059)	0.217 (0.010)	0.164 (0.001)	<.001
LDL-C, mmol/L						
Model 1	Reference	−0.052 (0.208)	−0.073 (0.126)	−0.081 (0.158)	−0.010 (0.773)	.328
Model 2	Reference	−0.058 (0.155)	−0.067 (0.156)	−0.082 (0.149)	0.017 (0.598)	.820
HDL-C, mmol/L						
Model 1	Reference	0.067 (<.0001)	0.095 (<.0001)	0.099 (<.0001)	0.198 (<.0001)	<.001
Model 2	Reference	0.068 (<.0001)	0.098 (<.0001)	0.099 (<.0001)	0.196 (<.0001)	<.001
Apo A1, mg/dL						
Model 1	Reference	8.635 (<.0001)	11.947 (<.0001)	12.591 (<.0001)	23.849 (<.0001)	<.001
Model 2	Reference	8.650 (<.0001)	11.950 (<.0001)	12.364 (<.0001)	23.386 (<.0001)	<.001
Apo B, mg/dL						
Model 1	Reference	0.008 (0.994)	0.356 (0.778)	−0.441 (0.772)	1.412 (0.107)	.167
Model 2	Reference	−0.177 (0.868)	0.345 (0.778)	−0.502 (0.733)	2.097 (0.015)	.037
Lp(a), mg/dL						
Model 1	Reference	−0.658 (0.411)	0.018 (0.984)	−1.686 (0.130)	−1.848 (0.004)	.003
Model 2	Reference	−0.739 (0.356)	−0.121 (0.896)	−1.794 (0.106)	−2.103 (0.001)	<.001
Non–HDL-C, mmol/L						
Model 1	Reference	0.003 (0.941)	0.001 (0.979)	−0.011 (0.864)	0.040 (0.280)	.367
Model 2	Reference	0.001 (0.990)	0.006 (0.916)	−0.010 (0.875)	0.072 (0.047)	.087
LDL-C/HDL-C						
Model 1	Reference	−0.155 (<.0001)	−0.201 (<.0001)	−0.220 (<.0001)	−0.270 (<.0001)	<.001
Model 2	Reference	−0.161 (<.0001)	−0.202 (<.0001)	−0.221 (<.0001)	−0.247 (<.0001)	<.001
TG/HDL-C						
Model 1	Reference	0.066 (0.309)	−0.002 (0.983)	0.081 (0.369)	−0.055 (0.288)	.513
Model 2	Reference	0.075 (0.222)	−0.009 (0.904)	0.092 (0.278)	−0.049 (0.321)	.575
Apo B/Apo A1						
Model 1	Reference	−0.045 (<.0001)	−0.055 (<.0001)	−0.064 (<.0001)	−0.086 (<.0001)	<.001
Model 2	Reference	−0.046 (<.0001)	−0.055 (<.0001)	−0.063 (<.0001)	−0.080 (<.0001)	<.001

Abbreviations: TG, total cholesterol; TC, triglyceride; LDL-C, low-density lipoprotein cholesterol; HDL-C, high-density lipoprotein cholesterol; Apo A1, apolipoprotein A1; Apo B, apolipoprotein B; Lp(a), lipoprotein(a); BMI, body mass index.
[a]Model 1: Age was adjusted.
[b]Model 2: BMI, education, cigarette smoking status, physical activity, hypertension, and diabetes were added.

Chinese population support the hypothesis that the inverse association between light to moderate alcohol consumption and CV risk may largely be explained by effects on HDL-C, and the harmful effects of heavy alcohol consumption are possibly related to an increase in TG and TC.[24]

In this population-based study among middle-aged Chinese populations, we found positive associations between alcohol consumption and level of HDL-C, apo A1, and TG in males. Our results also showed increased alcohol consumption associated with lower LDL-C/HDL-C and apo B/apo A1 in males. The results regarding TG and HDL-C are consistent with other epidemiological studies in Chinese, Koreans, Swiss, and Japanese populations.[23,25-27] In contrast, there are epidemiological studies like an Italian study of elderly individuals, which showed no significant association of HDL-C and TG with alcohol consumption.[28] This discrepancy may due to differences in dietary patterns, including the amount of total energy intake and percentage of fat intake, even differences in race/ethnicity in study populations. A recent meta-analysis also confirmed alcohol significantly increased the levels of HDL-C, but the association between TG and alcohol could not be found in this study.[29] However, previous studies also showed that the threshold alcohol intake of drinkers significantly lowered serum LDL-C, and prevalence of high serum LDL-C was lower in women and men.[26] A meta-analysis of the 27 prospective studies with at least 1 year of follow-up demonstrated a clear association between Lp(a) and CHD.[30] We found an association between alcohol drinking ≥30 g/d and Lp(a).

Previous studies considered the LDL-C–HDL-C ratio, TG–HDL-C ratio, lipid-related indices, and so on as predictors for CV risk.[13-15,31] Light to moderate drinking has been reported to show inverse associations with TG–HDL-C ratio and high LDL-C–HDL-C ratio in previous studies[32,33]; in our study, the inverse association was observed between LDL-C/HDL-C and apo B/apo A1 in males. A recent study showed the association of alcohol drinking with non-HDL cholesterol after adjustment

for age, body weight, and history of smoking in Austrians.[26] Serum apo A1 and HDL-C were significantly increased during 3 weeks of moderate alcohol consumption as compared with no alcohol consumption in the TNO Nutrition and Food Research study.[34] Our analysis failed to find this association, but the results of non-HDL-C and TG/HDL-C in males were similar to the Korea National Health and Nutrition Examination Survey (KNHANES) study.[23]

Our study had limitations. First, participants may have changed their drinking habits; they might have decided to consume less or no alcohol after they experienced some diseases. Second, alcohol consumption was derived from self-reporting, and heavy drinkers may have underreported their drinking habits. Third, the conditions of diet, which could confound the relationships among alcohol drinking and blood lipid levels, were not collected in the present study, and this may also cause a bias, although BMI was adjusted in the analysis, which reflects these factors to some extent. Additionally, different lipoprotein subfractions may have different effects on CV disease.[35] For example, independent association of small dense LDL with CV disease has been found in many epidemiologic or clinical intervention trials,[36] and another study also found that the protective effect of high HDL against CV disease was almost exclusively linked to the subfraction of large HDL2 particles but not to the small dense HDL3 subspecies.[37]

Male participants who consumed $\geq$30 g alcohol/d had significantly higher TC and lower Lp(a) compared with abstainers. Alcohol intake was positively associated with TG, HDL-C, and apo A1, and an inverse association was observed between total alcohol intake, LDL-C/HDL-C and apo B/apo A1 in males. Our results suggest that the beneficial effects of light to moderate alcohol consumption may be attributable to the higher HDL-C or apo A1 levels and decreased Lp(a), whereas the harmful effect of heavy alcohol consumption may be attributable to increased TG and TC. Because of the unknown risk of alcohol consumption, we cannot advise nondrinkers to drink for health benefit.

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Author's Note

Guang Hao and ZengWu Wang were the first authors. Guang Hao and ZengWu Wang contributed equally to the writing of this article. Zengwu Wang, Manlu Zhu, Linfeng Zhang, Zuo Chen, and Xin Wang designed the study. Guang Hao analyzed data and drafted the manuscript. Zengwu Wang critically revised and edited the manuscript. All authors contributed to discussion of manuscript.

Declaration of Conflicting Interests

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