Pharmacology Biochemistry and Behavior, Vol. 66, No. 1, pp. 163–167, 2000

0091-3057/00/$–see front matter

PII S0091-3057(00)00195-7

Blood Cholesterol Levels of 32-Year-Old Alcohol Consumers Are Better Than of Nonconsumers

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Received 19 October 1999; Revised 17 December 1999; Accepted 21 December 1999

KOPPES, L. L. J., J. W. R. TWISK, J. SNEL, W. VAN MECHELEN AND H. C. G. KEMPER. *Blood cholesterol levels of 32-year-old alcohol consumers are better than of nonconsumers.* PHARMACOL BIOCHEM BEHAV **66**(1) 163–167, 2000.—Blood cholesterol levels are expected to be important factors in the causal pathway between alcohol consumption and CHD. The relation between alcohol consumption and blood cholesterol levels is investigated in 130 men and 145 women aged 32.4 years old (±1.0), from the Amsterdam Growth and Health Longitudinal Study. When controlled for gender, cholesterol levels at age 13.1 years, and lifestyle at adult age (smoking, physical activity, dietary habits), no significant differences were found for total cholesterol (TC) levels between alcohol consumers and nonconsumers. Serum high-density lipoprotein (HDL) cholesterol levels were 0.12 mmol/l higher in subjects consuming ⩾100 grams of alcohol per week than in nonconsumers ($p < 0.05$). Regression coefficients of subjects consuming 10 to 50, or 50 to 100 g alcohol per week did not differ statistically from those of nonconsumers. The positive relation between alcohol consumption and serum HDL was modified by smoking (found in nonsmokers, but not in smokers). No differences between beer, wine, and spirits were found for their relation with serum HDL. In conclusion, 32.4-year-old nonsmoking subjects who consumed ⩾100 g of alcohol per week had improved HDL levels compared with nonconsumers, whereas the protective effect of drinking smaller amounts of alcohol did not reach statistical significance. © 2000 Elsevier Science Inc.

Alcohol Ethanol HDL Blood cholesterol Beer Wine Spirits Smoking

SEVERAL reviews of epidemiological studies report a protective effect of alcohol consumption on fatal and nonfatal coronary heart disease (CHD) (13,25,27,32,34,38). What the shape of this relation looks alike is still unclear. Rimm et al. (38), and Marmot and Brunner (27) found an inverse linear, Maclure an L-shaped (25), and Renaud et al. (34) found a U-shaped relation between the consumption of alcohol and CHD. These inconsistent findings may be related to the age and other CHD risk factors (7,21) of the subjects involved, the types of alcoholic beverages consumed (38), or the absence of high level consumers in the studies showing an inverse linear, L-, or U-shaped relation with CHD.

Serum cholesterol concentrations appear to play an important role in the relation between alcohol consumption and CHD. Alcohol increases the serum high-density lipoprotein (HDL) cholesterol (5,18,24,31,42), and may decrease the low-density lipoprotein (LDL) cholesterol concentrations in blood (5,10,18,24). Through this mechanism, about 50% of the decreased risk for CHD can be explained (5,24,40,42,47). Serum apolipoproteins A-1 and A-2 (4,18), lipoprotein(a) (15, 18,30,35), and fibrinogen (11,18,26,36,39) may be other factors in the causal pathway between alcohol consumption and CHD. They are assumed to be related to the unexplained 50%.

If the type of alcoholic beverage matters is the subject of debate (9,38). Some studies did not find a difference between beer, wine, and spirits in their relation with CHD or CHD risk factors (18–20,23,37,41,47). Others found a more beneficial effect of wine and/or beer than of spirits (8,21,22,45). This difference is often attributed to other protective substances than alcohol, like antioxidants and flavonoids, which are es-

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pecially found in red wine (6,12). It is also hypothesized that the difference could be attributed to a more advantageous lifestyle that wine and beer drinkers may have over consumers of spirits, that was not adjusted for (38,44,47). The lifestyle factors that may have interfered are smoking, physical activity, and dietary habits.

The consumption of alcohol has a protective effect on CHD at adult and old age. Whether it is protective before the age of 40 years is unknown. However, the process of arteriosclerosis starts at an early age, and alcohol is then likely to then decelerate the arteriosclerotic process. The present study investigates the linear relation between the consumption of alcohol and two important risk indicators for CHD: total, and HDL serum cholesterol. If the relation between alcohol consumption and cholesterol levels is modified by sex, smoking, physical activity, or dietary habits, and if the relation differs for the consumption of beer, wine, and spirits will be studied as well.

METHOD

Subjects and Design

The Amsterdam Growth and Health Longitudinal Study (approved by the medical ethical committee of the Vrije Universiteit, Amsterdam, The Netherlands) is an observational longitudinal study. It started in 1977, with 188 boys and 205 girls (mean age 13.1 years, standard deviation 0.7) from one secondary school in, and one near, the city of Amsterdam. The subjects were tested on a wide range of characteristics [see (16) and (17) for a more detailed description of the study]. Seventy percent (130 men and 145 women) was measured again in 1996 or 1997, at mean age 32.4 years (±1.0). The present analyses refer to the data gathered at mean age 32.4 years.

Measurements

Alcohol consumption at the mean age of 32.4 years was measured with a crosscheck dietary history interview covering the month prior to the interview (33). The average amount and number of alcoholic beverages consumed were converted into the amount of pure alcohol (in grams) that were consumed in a week. Alcoholic beverages were classified as "spirits" when the alcohol content was higher than 16 g per 100 g of the beverage, as "wine" when the beverage was not a "beer," and the alcohol content was less than 16 g (almost 95% true wine, 5% portified wine, <1% others). A distinction was made between "nonconsumers" [teetotalers and subjects consuming less than 10 g of alcohol (approximately one unit) per week], light drinkers (10 to 50 g/week), moderate drinkers (50 to 100 g/week), and "heavier" drinkers (⩾100 g/week).

Total serum cholesterol (TC), and serum HDL cholesterol were determined from venous blood taken between 0830 and 0930 h from the antecubital vein. TC was analyzed according to the method of Huang et al. (14) and Abell et al. (1). The serum concentration of HDL cholesterol was determined according to the Burstein and Samaille method (3).

Considered to be potential confounding factors are sex and adult-age smoking, physical activity, and dietary habits. From information obtained by questionnaire, subjects were characterized as nonsmokers, light smokers (1 to 10 cigarettes per day), or heavy smokers (⩾10 cigarettes per day). Daily physical activity was assessed by means of a standardized interview, inquiring about activities in the previous 3 months (28). Considering the intensity, performed activities were given a metabolic equivalent (MET) score (one MET corresponds to the metabolic rate while sitting at rest). Activities with an intensity of at least four MET were multiplied by the total weekly time spent doing them. The dietary habits that are considered as potential confounders are weekly averages of the total consumption of fat, the ratio between the intake of polyunsaturated and saturated fatty acids, and the consumption of cholesterol.

Data Analyses

Linear regression analyses were performed with TC and HDL measured at mean age 32.4 years as dependent variables, and total alcohol consumption as independent variable. Analyses were performed with "nonconsumers" as the reference category. In the first analyses was corrected for sex only. In subsequent analyses, TC or HDL levels that were obtained at mean age 13.1 years were forced into the model as a covariate. The addition of these prognostic factors results in an estimate of the relation between alcohol consumption at age 32.4 years, and the change that has occurred in cholesterol levels since age 13.1 years. Finally, current smoking status, physical activity, and dietary habits were entered into the model as covariates.

The full model was checked for possible effect modification by sex, smoking, physical activity, and dietary habits by addition of an interaction term. When the interaction term was statistically significant, additional stratified analyses were performed on the full model (in these analyses on interaction, a rather liberal significance level of $p < 0.10$ was used).

Differences between the full model regression coefficients for the separate beverages were tested with *t*-tests for the dependent variables showing a significant ($p < 0.05$) relation with total alcohol consumption. As the numbers of subjects with "heavier" beer, wine, or spirits drinking were rather small, a distinction was made between two drinking levels only, the lighter drinkers (10 to 70 g alcohol from the certain beverage per week), and the relatively "heavier" beer, wine, or spirits drinkers (⩾70 g/week).

TABLE 1

ALCOHOL CONSUMPTION AND BLOOD CHOLESTEROL CHARACTERISTICS OF THE SUBJECTS AT MEAN AGE 32.4 YEARS (±1.0)

	Men (n = 130)	Women (n = 145)
N drinking		
<10 g alcohol per week	16	57
10 to 50 g alcohol per week	34	36
50 to 100 g alcohol per week	29	31
⩾100 g alcohol per week	51	21
N drinking		
beer	97	36
wine	67	79
spirits	32	13
Mean alcohol intake (g/drinker/week)	117 (±112)	71 (±48)
Mean TC (mmol/liter)	5.01 (±0.97)	4.89 (±0.77)
Mean HDL (mmol/liter)	1.21 (±0.26)	1.59 (±0.34)

Values in parentheses are standard deviations.
TC: total cholesterol.
HDL: high-density lipoprotein cholesterol.

TABLE 2

REGRESSION COEFFICIENTS FOR TC LEVELS IN 32.4-YEAR-OLD LIGHT, MODERATE, AND "HEAVIER" DRINKERS COMPARED WITH "NONDRINKERS"

	Model 1		Model 2		Model 3	
	β	95% CI	β	95% CI	β	95% CI
<10 g/week (ref.)	0		0		0	
10–50 g/week	−0.01	−0.37; 0.22	−0.01	−0.32; 0.17	−0.10	−0.35; 0.15
50–100 g/week	−0.01	−0.39; 0.22	−0.01	−0.31; 0.20	−0.12	−0.39; 0.14
⩾100 g/week	0.13	−0.17; 0.44	0.16	−0.09; 0.42	0.13	−0.14; 0.39

Model 1: crude model, corrected for sex only.
Model 2: model 1 with correction for TC levels at age 13.1 years.
Model 3: model 2 with a correction for current smoking status, physical activity, and dietary habits.
95% CI: 95% confidence interval.

RESULTS

Alcohol consumption and blood cholesterol characteristics of the 130 male and 145 female subjects are shown in Table 1. More women (39%) than men (12%) abstain from alcohol, and a woman who drinks, consumes about 60% of the average amount of alcohol that the men do. In women, wine is the beverage of preference, while beer is in men. About 60% of the men and 30% of the women drink more than just one beverage. Prevalence of harmful drinking was low. Only 11% of the male and 6% of the female subjects consume more than the "maximal level of harmless drinking" as defined by the NEI [>210 or >140 g/week, respectively; (29)].

Table 2 shows the regression coefficients for the three drinking groups compared with the "nonconsumers" for TC, and Table 3 for HDL. No significant regression coefficients were found for TC. For HDL, the most beneficial levels were found in subjects with the highest level of alcohol consumption. Additional adjustment for, respectively, TC or HDL obtained at age 13.1 had an only marginal effect on the regression coefficients (model 2). Thus, the relation between alcohol consumption at age 32.4 is about equally related with cholesterol level at age 32.4 as with the change in cholesterol levels over the previous 19 years. Additional adjustment for the adult-age lifestyle variables (smoking, physical activity, and dietary habits) usually led to slightly larger regression coefficients (model 3), indicating that part of the relations was confounded by these variables.

The relation between alcohol consumption and HDL was modified by smoking, but not by sex, physical activity, or dietary habits. In nonsmokers, the relation was positive, while negative trends were found in smokers (Table 4). As only five smoking subjects abstained from alcohol, the confidence intervals in smokers are large.

Figure 1 shows the full model HDL regression coefficients for light and "heavier" beer, wine, and spirits consumption. No differences between the beverages were found (each $p > 0.10$).

DISCUSSION

The men and women in this study were from a general population having mean serum cholesterol levels, percentages of alcohol consumers, and levels of alcohol consumption that are comparable to those found in other surveys in general populations in The Netherlands (2,43). The purpose of the present study was to investigate the relation between the consumption of alcohol and two risk factors for coronary heart disease (CHD); the serum concentration of total cholesterol (TC) and high-density lipoprotein cholesterol (HDL). No significant relations were found with TC, while more favorable

TABLE 3

REGRESSION COEFFICIENTS FOR HDL LEVELS IN 32.4-YEAR-OLD LIGHT, MODERATE, AND "HEAVIER" DRINKERS COMPARED WITH "NONDRINKERS"

	Model 1		Model 2		Model 3	
	β	95% CI	β	95% CI	β	95% CI
<10 g/week (ref.)	0		0		0	
10–50 g/week	0.04	−0.04; 0.12	−0.01	−0.09; 0.08	0.02	−0.07; 0.10
50–100 g/week	−0.01	−0.09; 0.08	0.01	−0.08; 0.10	0.02	−0.07; 0.11
⩾100 g/week	0.08	−0.00; 0.16	0.09*	0.00; 0.18	0.12*	0.02; 0.21

Model 1: crude model, corrected for sex only.
Model 2: model 1 with correction for HDL levels at age 13.1 years.
Model 3: model 2 with a correction for current smoking status, physical activity, and dietary habits.
95% CI: 95% confidence interval.
*$p < 0.05$.

TABLE 4

REGRESSION COEFFICIENTS FOR HDL LEVELS IN 32.4-YEAR-OLD LIGHT, MODERATE, AND "HEAVIER" DRINKERS COMPARED WITH "NONDRINKERS" IN NONSMOKERS AND SMOKERS*

	Nonsmokers		Smokers	
	β	95% CI	β	95% CI
<10 g/week (ref.)	0		0	
10–50 g/week	0.03	−0.06; 0.13	−0.19	−0.48; 0.10
50–100 g/week	0.02	−0.08; 0.12	−0.19	−0.47; 0.10
⩾100 g/week	0.15†	0.05; 0.25	−0.12	−0.40; 0.16

*Analyses are corrected for sex, HDL levels at age 13.1 years, physical activity, and dietary habits.

†$p < 0.01$.

95% CI: 95% confidence interval.

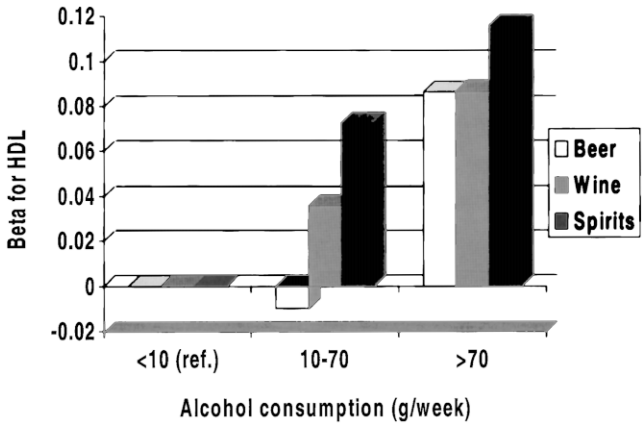

FIG. 1. Regression coefficients for HDL levels in 32.4-year-old light and "heavier" beer, wine, and spirits consumers compared with nonconsumers.

HDL levels were found for subjects who consumed at least 100 g of alcohol per week. The relation between alcohol and HDL appeared to be stronger after correction for baseline HDL levels, smoking, physical activity, and dietary habits. Other studies that did not control for these confounding variables extensively may, therefore, have underestimated the relation between alcohol and HDL.

The absence of a significant relation of alcohol consumption with TC, but presence of a positive relation with HDL, may be caused by low-density lipoprotein (LDL) cholesterols that are expected to be negatively related to alcohol consumption (5,10,18,24), but next to HDL are part of TC. Unfortunately, LDL cholesterols were not measured in the present study. As can be expected, the results for the ratio between TC and HDL were comparable to those of HDL: more favorable ratios in drinkers compared with nondrinkers, however, statistically significant at the highest level of consumption only (data not shown).

Smoking did not modify the relation between alcohol and CHD (40,46), no studies were found that had tested possible effect modification of smoking on the relation between alcohol and HDL. The interaction effect found in the present study may be related to behavioral differences between smokers and nonsmokers. Smokers may have a different pattern of alcohol consumption than nonsmokers; they may binge more often, or they may drink less often with meals. The interaction may also exist at a biochemical level, when a certain ingredient of cigarettes prevents the positive effect of alcohol on serum HDL.

The trend for, from a health perspective, better blood HDL levels with increasing alcohol consumption, should be interpreted with caution. The subjects in the "higher level of consumption group," in fact, were not very heavy consumers. Mean consumption in this group was 186 (±103) g of alcohol per week. According to the NEI (29), 68% of these subjects are "harmless drinkers." It should also be noted that blood cholesterol is just one indicator of a person's health status, and for several other indicators it is known that each increase in alcohol consumption has a negative effect. Also, most *p*-values in this study did not reach statistical significance, though the results suggest that alcohol is potentially protective for CHD at a relatively young year of age already.

In accordance with findings from the British Regional Heart Study (47), no differences between beer, wine, and spirits were found for their relation with HDL cholesterol. It, thus, appears that alcohol, and not any other substance of a certain beverage, has its effect on HDL.

To conclude, no relation was found between alcohol consumption and serum TC, while better serum HDL profiles were found for nonsmoking subjects who consumed at least 100 g of alcohol per week compared with nonsmoking alcohol abstainers. In subjects who consumed less than 100 g of alcohol per week, no differences with the nonconsumers were found. In smoking subjects, negative trends on HDL levels were found for alcohol consumption. Although the present finding is an important finding for public health, the overall risks of drinking alcohol in young persons probably, at least in the short term, outweigh the potential benefits. Therefore, young persons should probably not be advised to start drinking or to increase the amount of alcohol they consume to a level of at least 100 g per week.

ACKNOWLEDGEMENTS

This study was financially supported by the Dutch Heart Foundation (grant 76051-79051), the Dutch Prevention Fund (grants 28-189a, 28-1106, and 28-1106-1), the Dutch Ministry of Well Being and Public Health (grant 90-170), the Dairy Foundation on Nutrition and Health, the Dutch Olympic Committee/Netherlands Sports Federation, and Heineken Inc.

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