Alcohol Consumption and the Diet-Heart Controversy

By: Joseph J. Barboriak, ScD, Harvey W. Gruchow, PhD, and Alfred J. Anderson, MS

Abstract:

The effectiveness of dietary changes as a means of reducing blood lipid levels and ultimately controlling the development of coronary heart disease has been debated for many years. The possible effects of alcohol consumption on blood lipids were usually not considered. Our findings indicate a significant positive correlation between the extent of coronary artery occlusion and total plasma cholesterol levels and a negative association between the coronary occlusion and high density lipoprotein (HDL) cholesterol. Since moderate alcohol consumption increases the HDL cholesterol levels, one can also postulate that it affects coronary artery lesions. The attenuating effect of alcohol on the coronary occlusion was negated by sporadic drinking of large amounts of alcohol. In evaluating the possible effect of alcohol on coronary artery disease, it is also necessary to consider Its addictive potential as well as other untoward sequelae of alcohol consumption such as hypertension, damage to the myocardium, and increased prevalence of malignancies.

Article:

DESPITE the recent remarkable reduction in the mortality rates from cardiovascular diseases,[1] this category continues to be the leading cause of death in the United States, accounting for almost 40% of the deaths in 1978.[2] Epidemiological studies have helped to identify several factors believed to be involved in the development of cardiovascular lesions.[3,4] Some of the factors such as age, sex, or heredity are beyond effective control; others such as smoking, increased blood pressure, or increased blood lipid levels are more amenable to change.

The extent to which the last factor, i.e., blood lipids, can or should be modified by dietary means in order to reduce the prevalence of coronary heart disease is the subject of a spirited debate, also known as the diet-heart controversy.[5,6] The suggested main dietary modifications include the reduction of total calories derived from fat, an increase in the ratio of polyunsaturated to saturated fatty acids, and a education of dietary cholesterol. Consumption of alcohol, a relatively common source of dietary calories, has only been mentioned in very general terms.

Yet, an association between alcohol consumption and changes in plasma lipid levels, the presumed mediators of dietary intervention, has been known since the early studies of this interaction. A number of clinical investigations[7-10] have described an enhancing effect of alcohol on blood lipids, especially the plasma triglycerides.

Additional studies have shown that this increase in plasma triglycerides can occur after ingestion of relatively small amounts of alcohol, especially in subjects with increased levels of very low density lipoprotein (VLDL).[11, 12] Due to the relatively easy and reproducible elevation of plasma triglyceride levels following ethanol ingestion and their clear association with VLDL, little attention has been given to the possible effect of ethanol on other lipoprotein classes. However, sporadic reports in the literature have indicated that ingestion of larger amounts of alcohol may be associated with a rise of high density lipoprotein (HDL) cholesterol and a moderate reduction of low density lipoproteins (LDL).[13-16] A slight to moderate rise of HDL was described by Johansson and Laurell[13] in chronic alcoholics. In animal studies, Baraona and Lieber[17] observed that chronic,

but not acute, administration of alcohol led to an increase in HDL, In a more recent study, Belfrage et al.[14] administered an equivalent of 75 g of absolute alcohol, in divided doses, daily for 5 weeks to young healthy volunteers and reported a significant rise in HDL-cholesterol levels after 3 weeks of alcohol consumption. This confirmed the earlier observation[17] that the HDL increase following ingestion of alcohol is seen only after repeated alcohol doses.

The possible association between alcohol consumption and increased HDL-cholesterol levels is of interest since it is believed that FIDL-cholesterol reduces the development of atherosclerosis. A number of studies have found that the presence of high HDL-cholesterol levels in both men and women indicates less extensive coronary heart disease. The data from the Cooperative Lipoprotein Phenotyping Study[18] suggest that low HDL-cholesterol values may be more predictive or indicative of the development of coronary artery disease than any other risk factor. The HDL-cholesterol levels are increased in women,[19] and in physically active individuals.[20] Both groups have a lower incidence of coronary artery disease than their counterparts, i.e., men in general or physically inactive individuals.

In order to place the relationship between alcohol consumption and HDL levels in proper perspective, one should also consider the extent of drinking in the United States. Recent computation of the Health Interview Survey, the Health and Nutrition Examination Survey of the Opinion Research Corporation, has indicated that 25-30% of the over 50,000 surveyed individuals consumed more than 35 drinks per month, and approximately 10% consumed 14 or more drinks per week. Our survey of over 3,000 patients with some symptoms of coronary heart disease has shown that almost 80% of the respondents reported some alcohol intake.[22] It would seem, therefore, that a considerable pro-portion of the population is exposed to the lipid-modifying effect of alcohol.

In order to investigate the relationship between alcohol intake and plasma lipids in greater detail and on a more quantitative basis, we have obtained information on these two variables in a population of men and women who have had coronary arteriography because of symptoms of coronary artery disease. In this procedure, a radiopaque dye is injected into the coronary artery, an x-ray picture of the heart is then taken and one can see the location and extent of the occlusion in the individual arterial branches. The degree of occlusion may then be expressed as an occlusion score,[23] and correlated with both an alcohol intake and the level of plasma lipids. An occlusion score of 300 indicates that all three main coronary branches are occluded, 0 indicates no occlusion. Each patient also received a questionnaire asking for the number of alcoholic drinks consumed during an average week. The data were then transformed into ounces of absolute alcohol. Administration of the same questionnaire to 400 consecutive patients 2-4 weeks after the first inquiry has indicated considerable stability of drinking habits in this population ($r = 0.85$).

The extent of the association between the total plasma cholesterol level and the coronary artery occlusion in 3,587 men and 961 women is shown in Figs. 1 and 2. The data, corrected for confounding variables, are shown separately for two different age groups. They indicate an almost linear relationship between the change of plasma total cholesterol level and the extent of coronary artery occlusion. Therefore, if one assumes cause-effect interaction between these two variables, correction of the high plasma total cholesterol levels should lead to less pronounced coronary lesions, justifying proper intervention to modify the plasma cholesterol levels.

Fig. 3 illustrates the relationship between plasma cholesterol levels and the extent of coronary artery disease. The higher plasma HDL-cholesterol levels were associated with lower occlusion scores.

Information on the relationship between alcohol intake and the levels of high density lipoprotein cholesterol is shown in Fig. 4. There was almost a linear increase in the reported alcohol intake as the plasma HDL-cholesterol levels of the male patients increased from less than 40 to over 50 mg/dl. The difference was statistically significant *(p < 0.01).*
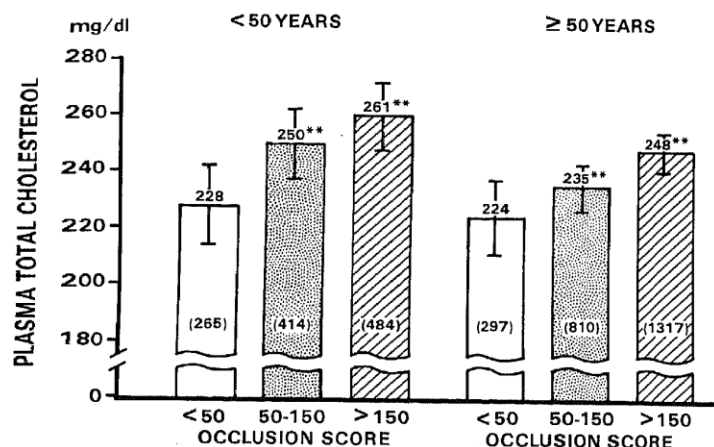
Figure 1. Association between plasma total cholesterol levels and angiographically ascertained coronary artery occlusion in 3,587 male heart patients. Vertical lines represent ± SD. ** = $p < 0.01$.
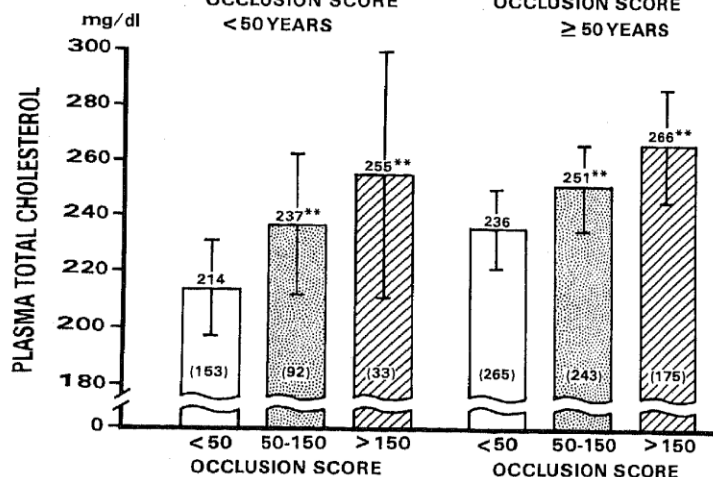

Figure 2. Association between plasma total cholesterol levels and coronary artery occlusion in 961 female heart patients.

Proper caution should be exercised in considering these findings. The antiocclusive effect of alcohol seems specific for coronary arteries and has not been observed for the cerebral blood vessels.[24] Furthermore, excessive alcohol consumption can lead to an enlarged heart, to structural changes in the heart muscle, and impairment of heart contractions.[25-27] In addition, our more recent data indicate that even the association between coronary artery occlusion and alcohol consumption may be more complicated than originally anticipated.[28]

We have recently collected information on the drinking pattern, i.e., whether individuals consume alcohol at regular intervals or whether they tend to consume the drinks only occasionally, i.e., in a "binge" drinking pattern. Our experience has suggested that most drinkers consume alcoholic beverages at regular intervals and, in addition, they sporadically tend to drink outside of the regular drinking pattern. In our study, most drinkers have consumed alcohol on an average of every 1.5 days, the average amount being approximately 45 ml. This represents about 1.5 oz of absolute alcohol. Most of these individuals also consumed alcohol sporadically, in average periods of about 12 days. At this time, they imbibed an equivalent of about 82 ml or close to 3 oz of absolute alcohol.

Table 1. Relationship of Sporadic Drinking Ratio to Occlusion Score, Total Alcohol Consumption, and Age*

	Sporadic drinking ratio (SDR)†				
	1.00–1.49	1.50–1.99	2.00–2.99	3.00+	p value
Number of patients	72	101	89	29	
Occlusion scores	146.0 ± 9.5	141.0 ± 8.3	157.0 ± 8.2	177.0 ± 7.9	0.03
Weekly alcohol consumption (ml)	345.2 ± 44.8	321.6 ± 34.0	289.1 ± 29.5	236.0 ± 26.4	0.09
Age	53.4 ± 1.0	54.2 ± 0.8	52.5 ± 0.8	52.7 ± 1.3	0.29

* Values given are means ± SE; p values are for linear associations with SDR, based on F ratios.
† Minimum SDR = 1.0.

The ratio of the amount of alcohol consumed during the "special" occasions divided by the amounts taken during the "regular" drinking days provides information on the extent of the sporadic drinking (sporadic

drinking ratio (SDR)). Table 1 shows the association between the extent of coronary artery occlusion and SDR. This finding suggests that an increase in the amount of alcohol consumed outside of the regular drinking pattern (higher SDR) is connected with more coronary occlusion. Interestingly, this effect of SDR on coronary artery occlusion does not seem to depend on the total amount of alcohol consumed. The definitive explanation on the mechanism(s) by which the SDR pattern affects coronary artery lesions is not available at this time. However, in preliminary studies, we have observed that when alcohol is consumed in a high SDR pattern, the plasma HDL-cholesterol levels do not increase as observed previously. The effect of the high SDR pattern on plasma HDL-cholesterol levels may be opposite that of the influence of moderate alcohol consumption and this may have affected the extent of coronary artery occlusion.

In conclusion, our findings have shown a marked positive association between the levels of plasma total cholesterol and the extent of coronary artery occlusion, and a negative relationship between the levels of HDL-cholesterol and the coronary occlusion. This suggests that measures modifying concentrations of these plasma lipid classes may affect the severity of the coronary lesions. Since moderate alcohol consumption leads to an increase in plasma HDL-cholesterol and less extensive coronary occlusion, this relatively common dietary component should be considered in evaluating the role of diet in the development of coronary artery disease. However, in considering the association between alcohol intake and coronary artery disease, attention should also be given to the untoward effects of the sporadic drinking pattern,[28] the addictive potential of alcohol, its hypertensive effects[29] predisposing to stroke,[30] as well as to a higher prevalence of malignancies in drinkers of alcoholic beverages.[31] It is clear that increased alcohol consumption should not be viewed as a possible means of preventing or reducing coronary artery disease. More efficient control of smoking and hypertension, reduction of levels of atherogenic low density lipoproteins by dietary or pharmacologic treatment, as well as increased physical activity represent the preferred approaches in achieving this goal.
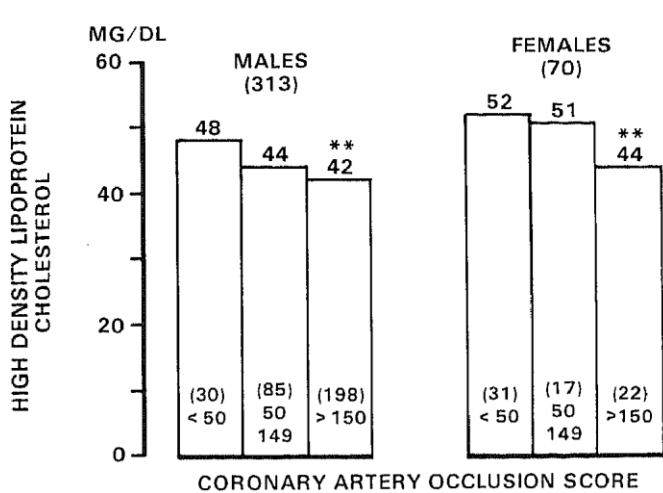
Fig. 3. Inverse relationship between HDL-cholesterol level and the extent of coronary artery occlusion in 313 male and 70 female heart patients.
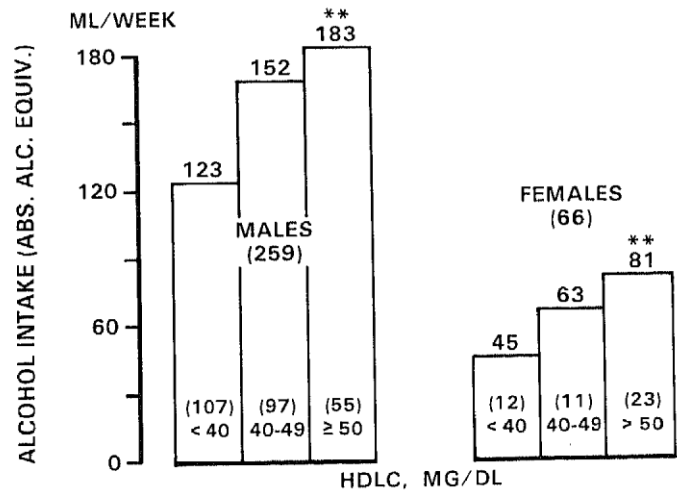
Fig. 4. Association between alcohol intake and plasma HDL-cholesterol levels in male and female heart patients.

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