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Alcohol-Induced Changes in Lipids and Lipoproteins

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Fasting plasma samples from 42 voluntary subjects of moderate to high socioeconomic status families and living in Delhi, taking 5–20 oz/week of alcohol regularly, and 42 normal subjects from comparable socioeconomic status with no history of intake of alcohol and no evidence of any known metabolic or coronary problems were analyzed for cholesterol and triglycerides and their levels in lipoprotein fractions, i.e., very low density lipoproteins (VLDL), low density lipoproteins (LDL), and high density lipoproteins (HDL). Free fatty acids, urea, uric acid, and glucose levels were also determined and dietary intake was calculated in the two groups.

Cholesterol and triglycerides in plasma, and VLDL and LDL fractions were found to be increased in the alcohol-taking group even though their intake of alcohol was moderate. VLDL fraction formed 65 and 32% of total triglycerides and cholesterol, respectively. Sugar, uric acid, and free fatty acids (FFA) increased significantly in the alcohol-taking group. Total energy intake was significantly higher in the alcohol-taking group mainly from significant high intake of proteins and fats. Calculated on the basis of intake/day/kg of body weight, the difference in intake of protein, fat, and carbohydrates was insignificant. Regular intake of even small quantity of alcohol along with food rich in proteins and fats may induce elevation of high cholesterol-containing lipoproteins, i.e., LDL and VLDL with increased hypertriglyceridemia, and these increased levels may constitute important coronary risk factors.

IT IS well established now that ethanol produces a consistent and rapid increase in serum lipids in normal subjects. The most affected constituent is triglycerides[1] which have been known to be one of the risk factors of ischemic heart disease (IHD).[2] The administration of ethanol in combination with fat-containing meal significantly enhances the mechanism of increased hepatic production or removal of serum lipoproteins, mainly, very low density lipoproteins (VLDL).[3,4] The effect of ethanol on the free fatty acids (FAA) is dose related. Whereas, moderate dose results in low concentration of plasma FFA of short duration and a reduction in circulating glycerol,[1,5] a high dose results in significant increases in plasma FFA.[6] Besides, chronic alcohol consumption increases serum cholesterol concentration in man.[1,6] This may induce the risk of IHD which is related to high cholesterol-containing lipoproteins, i.e., low density lipoproteins (LDL) and VLDL. The increased secretion of VLDL has also been attributed to alcohol-induced elevation of FFA levels in plasma which is derived primarily from the diet with constant high doses of alcohol.[7]

Several studies done in Western societies have shown a close relationship between alcohol and increased levels of lipids and their fractions in serum. Since no such studies are available in India, where alcoholism is not a major social problem as in the Western countries and also the type of food and eating habits are entirely different, it was assumed that consumption of alcohol along with sufficient intake of food may induce changes in blood lipids and lipoproteins based on local environmental conditions. In this paper, data are presented on lipids and lipoprotein composition of an alcohol-taking group when compared to age and socioeconomic status matched group in relation to intake of nutrients and other biochemical factors.

MATERIALS AND METHODS

Fasting blood samples were drawn from 42 voluntary subjects living in different parts of Delhi, from moderate to high socioeconomic status families, taking regularly between 5 and 20 oz of alcohol/week. These subjects were taking alcohol for periods varying from 6 months to over 6 years. Ethanol consumption per day/kg of body weight calculated on the basis of ethanol consumption and average body weight showed that these alcohol-taking subjects were moderate drinkers. There was no evidence of any liver disease in these subjects as judged by normal liver function tests.

Fasting blood samples were also drawn from 42 normal healthy volunteers who were comparable to the alcohol-taking group in age and socioeconomic status. In these control subjects there was no known manifestation or evidence of any metabolic disease. Their fasting plasma glucose was in the range of 70–100 mg/100 ml and there was no evidence of hypertension or any coronary problems. Their weight was within 20% of desirable weight for that age group in reference to the height weight chart of Life Insurance Corporation of India. These normal controls did not give a history of intake of alcohol, except two patients who had consumed alcohol less than 2–3 oz/year.

The diet history was taken from all the alcohol-taking subjects and control subjects by 24 hr recall method and calculated for various nutrients, i.e., carbohydrates, fats, proteins, and total energy intake. The height and weight were recorded.

Blood after 12–14 hr of overnight fasting was drawn in 1 mg/ml EDTA-disodium salt for lipids and lipoprotein estimation and in plain vials for urea, uric acid, sugar, and FFA. The samples were stored at 4°C and processed within 48 hr of collection.

Quantification of lipoprotein fractions was carried out by the dual precipitation technique used by Wilson and Spiger.[8] Cholesterol and triglycerides were determined in plasma, HDL, and HDL + LDL fractions by the method of Chiamori and Henry[9] and Van Handel and Zilversmit,[10] respectively. The values for VLDL were obtained by substraction. Urea,[11] uric acid,[12] and sugar[13] in serum were determined by standard techniques. Nonestesified fatty acid (NEFA) was determined by the technique of Novak.[14]

RESULTS

Mean age of 36.7 ± 9.5 years and height of 170.7 ± 6.6 cm in the control group was well comparable to the mean

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age of 39.4 ± 11.8 years and height of 172.0 ± 6.9 cm in the alcohol-taking group (Table 1). In the alcohol-taking group the mean weight was 72.5 ± 8.2 kg which was significantly higher than the control group with mean weight of 64.6 ± 7.3 kg. In terms of per cent of weight to expected weight, the values were 113.5 ± 15.7 and 103.1 ± 9.1%, respectively, in the alcohol-taking group and the control group.

Table 1. Height, Weight, and Values of Nutrients in Control and Alcohol-Taking Group

Groups	Age (years)	Height (cm)	Weight (kg)	Weight (%)
Alcohol				
(n = 42)				
Mean ± SD	39.4 ± 11.8	172 ± 7	73 ± 8	114 ± 16
Control				
(n = 42)				
Mean ± SD	36.7 ± 9.5	171 ± 7	65 ± 7	103 ± 9
p =	NS*	NS*	<0.001†	<0.001†

* NS, not significant.
† p value of <0.001 shows that the difference was significant at 99.9% level.

Table 2. Values of Nutrients Intake in Control and Alcohol-Taking Group

Group	Energy (kcal)	Carbohydrate (g)	Proteins (g)	Fats (g)
Alcohol				
n = 42				
Mean ± SD	2470 ± 547*	340 ± 75	84 ± 27	86 ± 24
Percentage	100	55.1	13.6	31.3
Control				
n = 42				
Mean ± SD	2221 ± 406	321 ± 75	70 ± 14	73 ± 27
Percentage	100	57.8	12.6	29.6
p =	<0.05†	NS‡	<0.01†	<0.05†

* Shows that caloric values of alcohol is not added.
† p values of <0.01 and <0.05 show that the difference was significant at 99 and 95% level, respectively.
‡ NS, not significant.

Table 3. Nutrients Intake Per Day Per Kg of Body Weight

Groups	Carbohydrate (g)	Protein (g)	Fat (g)
Alcohol (n = 42)	4.79 ± 1.33	1.20 ± 0.45	1.17 ± 0.44
Control n = 42	4.81 ± 1.29	1.08 ± 0.30	1.11 ± 0.42
p =	NS*	NS*	NS*

Values shown mean ± SD.
* NS, not significant.

In the alcohol-taking group, the energy intake of mean 2470 ± 547 calories was from 340 ± 75 g of carbohydrates, 84 ± 27 g of proteins, and 86 ± 24 g of fats (Table 2), whereas in the control group the energy intake of mean 2,221 ± 406 calories was from 321 ± 75 g of carbohydrates, 70 ± 14 g of proteins, and 73 ± 27 g of fats. The caloric values of alcohol intake are not included in the present alcohol-taking group values because the quantities are approximations and not the measured quantities. The intake of calories was significantly higher ($p < 0.05$) in the alcohol-taking group. In this group there was significantly higher intake of proteins and fats. In terms of percentage, 55.1, 13.6, and 31.3% of the caloric intake was from carbohydrates, proteins, and fats, respectively, in the alcohol-taking group and the corresponding values in control group were 57.8, 12.6, and 29.6%, respectively.

Table 3 shows the values of nutrients intake per day/kg of body weight in both the control and the alcohol-taking groups. Among the alcohol-taking group 88% of subjects were nonvegetarian and 55% were smokers, whereas in the control group the percentage of nonvegetarians and smokers was 71 and 36%, respectively.

In none of the subjects was there any evidence of liver damage as observed from liver function tests. The values of lipids and their lipoproteins for both groups, i.e., the control and alcohol-taking groups, are presented in Table 4. In the alcohol-taking subjects, the values of total cholesterol and cholesterol in VLDL, LDL, and HDL fractions of lipoproteins were 208 ± 47, 66 ± 33, 111 ± 45, and 32 ± 12 mg/100 ml, respectively, whereas in the control group the values for the same fractions were 159 ± 26, 37 ± 17, 88 ± 25, and 33 ± 12 mg/100 ml, respectively. The values of triglycerides in plasma and its lipoprotein fractions, i.e., VLDL, LDL, and HDL, were 319 ± 136, 208 ± 124, 60 ± 31, and 52 ± 24 mg/100 ml, respectively, in the alcohol-takling group and 127 ± 30, 45 ± 24, 35 ± 19, and 48 ± 19 mg/100 ml, respectively, in the control group. The difference between the two groups was significant for cholesterol and triglycerides contents in plasma, VLDL, and LDL and nonsignificant for HDL.

The values of urea, uric acid, and sugar found in the alcohol-taking group were 14 ± 4, 4.5 ± 1.0, and 93 ± 19

Table 4. Cholesterol and Triglycerides Values in Plasma and Its Lipoprotein Fractions

	Cholesterol (mg/100 ml)				Triglycerides (mg/100 ml)			
Groups	Total C	VLDL C	LDL C	HDL C	Total TG	VLDL TG	LDL TG	HDL TG
Alcohol								
n = 42								
Mean ± SD	209 ± 47	66 ± 33	111 ± 45	32 ± 12	320 ± 136	208 ± 124	60 ± 31	52 ± 24
Percentage	100	32	53	15	100	65	19	16
Control								
n = 42								
Mean ± SD	158 ± 26	37 ± 17	88 ± 25	33 ± 12	128 ± 30	45 ± 24	35 ± 19	48 ± 19
Percentage	100	23	56	21	100	35	29	38
p =	<0.001	<0.001	<0.01	NS*	<0.001	<0.001	<0.001	NS*

Using Student's t test the p value of <0.001 and <0.01 shows that values were significantly higher at 99.9 and 99% level, respectively.
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age of 39.4 ± 11.8 years and height of 172.0 ± 6.9 cm in the alcohol-taking group (Table 1). In the alcohol-taking group the mean weight was 72.5 ± 8.2 kg which was significantly higher than the control group with mean weight of 64.6 ± 7.3 kg. In terms of per cent of weight to expected weight, the values were 113.5 ± 15.7 and 103.1 ± 9.1%, respectively, in the alcohol-taking group and the control group.

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Percentage	100	23	56	21	100	35	29	38
p =	<0.001	<0.001	<0.01	NS*	<0.001	<0.001	<0.001	NS*

Using Student's t test the p value of <0.001 and <0.01 shows that values were significantly higher at 99.9 and 99% level, respectively.

* NS, not significant.

Table 5. Values of Sugar, Urea, Uric Acid, and Free Fatty Acids in the Control and Alcohol Group

Groups	Sugar mg/100 ml	Urea mg/100 ml	Uric acid mg/100 ml	FFA meq/liter
Alcohol				
$n = 42$				
Mean ± SD	93 ± 19	14 ± 4	4.5 ± 1.0	1.06 ± 0.57
Control				
$n = 42$				
Mean ± SD	84 ± 11	14 ± 4	5.0 ± 0.8	0.67 ± 0.16
p value*	<0.001	NS	<0.02	<0.001

* p value of <0.001, <0.01, and <0.02 shows that the difference was significant at 99.9, 99, 98% level, respectively.

mg/100 ml, respectively, in comparison to control group values of 14 ± 4, 5.0 ± 0.8, and 84 ± 11 mg/100 ml, respectively (Table 5). The difference was significant for uric acid and sugar only. Free fatty acid value of 1.06 ± 0.57 meq/liter in the alcohol-taking group was significantly higher than the control group with mean values of 0.67 ± 0.16 meq/liter.

DISCUSSION

In the control subjects the values of lipids and its lipoprotein fractions were comparable to our earlier reported values[15] for North Indian subjects. The mean value 0.67 ± 0.16 meq/liter of free fatty acids was within normal reported value.[16]

In the alcohol-taking group total cholesterol and triglycerides in plasma and its VLDL and LDL fraction was found to increase significantly when compared to the control group whereas HDL fraction did not show any change. VLDL fraction formed 65 and 32% of triglycerides and cholesterol, respectively.

Changes in lipids and its lipoprotein fractions may be attributed to factors like socioeconomic status, diet intake, smoking, and type of food etc., in addition to hereditary factors. Both the groups, i.e., control subjects and subjects taking alcohol, were from a similar socioeconomic status. Fifty-five per cent of alcoholics were smokers as compared to 36% in the control group. Alcoholism has been found to be associated with smoking.[17] In the present alcohol taking group, 88% were nonvegetarians with a high intake of proteins and fats from animal sources. Their intake of total energy was high in comparison to control group. It seemed that they tended to eat more with drinks. Since no significant difference has been shown between vegetarians and nonvegetarians[18] from South India, no efforts were made to separate vegetarians from nonvegetarians in our study.

Diet, however, seems to play a major role in subjects taking alcohol. Alcohol consumption, along with high caloric diet, has been found to increase the cholesterol concentration in man and has been suggested to induce the risk of atherosclerotic vascular disease which is related to high cholesterol-containing lipoproteins, i.e., LDL and VLDL.[1,6] The significant high intake of fat along with high intake of energy may account for the significant increase in VLDL contents by enhancing the mechanism of increased hepatic production of or removal of lipoproteins.[3] Ethanol enhances the incorporation of chylomicrons fatty acids into newly synthesized VLDL.[19]

Blood HDL has been shown to be increased after chronic moderate intake of alcohol.[20–24] However, in the present study we failed to observe any significant increase in HDL cholesterol levels in the alcohol-drinking group. This may be partially due to a higher percentage of smokers in the alcohol-taking group. Smoking is associated with decreased HDL[25] levels due to an unknown mechanism,[26] thus smoking might have offset the stimulatory effects of alcohol on HDL levels.

The changes in lipids and lipoproteins of the alcohol-taking group in relation to diet does not seem to be carbohydrate-induced. The intake of carbohydrates between two groups was not significant. The difference could be due to high intake of fats and proteins or alcohol ingestion. However, based on nutrient intake per day/kg of body weight, no significant difference was found for any of the nutrients between the control and alcohol-taking group (Table 3).

The most affected constituent in lipids after intake of alcohol has been the triglycerides. There was a significant rise in the levels of triglycerides in the alcohol-taking group and out of which two-thirds was in the VLDL fraction only as against one-third in the VLDL fraction of the control group. The daily use of moderate amounts of alcohol seems to exacerbate hypertriglyceridemia.[27] Also, there was a significant increase in the LDL fraction of triglycerides and free fatty acids in the alcoholic group over the control group. Chait et al.[28] suggested that the proneness to the triglyceride's elevating effect of alcohol may be due to a relatively limited capacity for removal of triglycerides from plasma, especially due to associated diabetes and results both in accumulation of this lipid in the liver and in enhanced synthesis of triglycerides-rich lipoproteins.

Free fatty acids have been found to vary with the amount of alcohol consumed. In high doses of alcohol a significant increase in plasma concentration of FFA has been reported.[6] A significant increase of FFA in the alcohol-taking group, i.e., 1.06 meq/liter over the control group 0.67 meq/liter may be due to recirculation of dietary fatty acids or due to a constant high dose of alcohol intake. Elevation of FFA levels in plasma may be responsible for a higher secretion of VLDL.

In the alcohol-taking group (Table 5), the levels of glucose were found to be significantly higher than the control group. Moderate doses of alcohol have already been shown to impair glucose tolerance significantly.[29] In contrast, Ginsberg et al.[27] did not show any alterations in plasma glucose.

There was no significant difference between the two groups for content of urea. Since there was no evidence of any renal failure in the alcoholic group, it was presumed that the hypertriglyceridemic state was not due to a renal problem but was alcohol-induced only. Uric acid in the

alcoholic group was found to decrease significantly in comparison to the control group.

The study leads us to conclude the following.

1. Regular intake of even small amounts of alcohol may lead to changes in lipids and lipoproteins and may constitute important coronary risk factors.

2. Intake of alcohol associated with a diet rich in proteins and fats elevates cholesterol-containing lipoproteins, i.e., VLDL and LDL.

3. Based on per day per kg of body weight consumption of nutrients, the changes in lipids and their lipoproteins were alcohol-induced.

4. In our sample it was observed that moderate alcohol-taking subjects tended to eat more, especially animal fats and proteins.

5. There are indications that alcohol may lead to impaired glucose tolerance.

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