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EFFECT OF MILD, MODERATE AND HEAVY SMOKING ON HIGH DENSITY LIPOPROTEIN CHOLESTEROL (HDL-C)

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ABSTRACT

There are various environmental factors which alter the normal lipid profile. Smoking is one of them and play a very important role in alteration of normal lipid profile. Cigarette smokers have a high risk of developing several chronic disorders. These include several type of cancers, Chronic obstructive pulmonary disease and atherosclerosis which is a major contributor to the high number of deaths from smoking. Smoking causes harmful effects of total cholesterol and reduces the cardioprotective properties of high density lipoprotein cholesterol which increase the risk of coronary heart disease. Decreased serum high density lipoprotein cholesterol level is very common in the subjects with coronary heart disease. The serum high density lipoprotein cholesterol levels are usually lower in smokers as compared to non- smokers. However most of the studies have not showed that whether mild, moderate or heavy smoking affects the levels of high density lipoprotein cholesterol. In light of above the aim of the present study was to evaluate the effect of smoking on the levels of high density lipoprotein cholesterol in mild, moderate and heavy male smokers. This study enrolled 80 healthy male subjects, 20 as control, 20 mild smokers, 20 moderate smokers and 20 heavy smokers. All the subjects included in the study were between 30- 40 years of age group. The mean age ± SD was 36.3 ± 2.05, 37.1 ± 1.11, 36.25 ± 1.06 &36.70 ± 2.01 for controls, mild smokers, moderate smokers and heavy smokers respectively. The mean body mass index (BMI) ± SD was 21.91 ± 0.98, 21.94 ± 1.13, 21.86 ± 1.07 & 22.03 ± 1.13 for controls, mild smokers, moderate smokers and heavy smokers respectively. The present study showed significant difference in the levels of high density lipoprotein cholesterol in all groups. The mean ± SD value of HDL-C was 49.96 ± 3.96, 37.32 ± 3.26, 33.40 ± 1.65 and 31.71 ± 2.48 for control, mild smokers, moderate smokers and heavy smokers respectively. HDL-C was measured by Phosphotungtic Acid Method. Smoking whether mild, moderate, heavy changes the levels of HDL-C by decreasing its concentration in serum and hence increases the risk of coronary heart disease.

KEYWORDS: Cigarette, Smokers, High density lipoprotein cholesterol.

INTRODUCTION

Smoking is one of the addictive habits which is increasing sharply throughout the developing world and is one of the biggest threats to current and future world health. Smoking is one of the environmental factors responsible for atherosclerotic cerebrovascular and cardiovascular disease. Cigarette smoking is associated with increased risk of a various medical disorders. Many studies show the evidence that tobacco is closely linked with alteration of the normal levels of the lipid profile (1,2).

Smoking changes the blood lipid profile. Serum high density lipoprotein cholesterol and low density lipoprotein cholesterol levels have been reported to be lower and higher, respectively, in smokers than in non-smokers (2,3). Thus smoking may exert its atherogenic effects through blood lipid levels (4,5)

There are various mechanisms which lead to alteration in lipid profile by smoking are:

(a) Nicotine causes an increase in secretion of hepatic free fatty acids & triglyceride accompanied very low density lipoprotein cholesterol in the blood stream by increasing the secretion of catecholamines & thus stimulating sympathetic adrenal system resulting in increased lipolysis (6).

(b) Consumption of a diet lacking fiber and cereal content but enriched fat & cholesterol (7).

(c) Smoking is linked with increased level of plasma homocysteine which cause oxidative modification of low density lipoprotein cholesterol & decreases high density lipoprotein cholesterol (8,9).

High density lipoprotein (HDL) is a plasma lipoprotein transporting 30% phospholipid, 25% cholesterol and 5% triacylglycerol. Its high density is due to 40%

protein. The cholesterol associated with it is unutilized excess cholesterol from tissues. This cholesterol is returned to the liver. The liver converts it into bile acids, bile salts and cholesteryl esters. The two bile salts formed in the liver are sodium glycocholate and sodium taurocholate. These compounds are secreted by liver into bile and through bile they reach the small intestine. The returning of the scavenged cholesterol to liver from peripheral tissue by HDL is known as "Reverse Cholesterol transport" (10).

A higher level of HDL-C is an index of safety from the risk of CHD. This is due to the fact that unutilized excess cholesterol which is potentially harmful, is scavenged from the tissue by HDL preventing its deposition and plaque formation (11). Low plasma HDL-C is a risk factor for CHD particularly in the males. Experimental studies in laboratory animals and epidemiological studied in population groups have shown that raising the levels of HDL-C in plasma may retard the development of atherosclerosis (11,12).

Studies suggest that HDL-C levels are lower in smokers as compared to non-smokers However no one have been studied whether mild, moderate or heavy smoking affects the levels of high density lipoprotein cholesterol. In light of above the aim of the present study was to assess the effect of mild, moderate and heavy smoking on the levels of high density lipoprotein cholesterol.

MATERIAL AND METHODS

This study was carried out at Department of Biochemistry, Eras Lucknow Medical College &Hospital, Era University, Lucknow, (UP), India, over a period of six months, after permission of Ethics Committee.

The study included 80 healthy male subjects who were similar in age group (30-40 years). 20 mild smokers (those who smoked less than10 cigarettes per day), 20 moderate smokers (those who smoked more than 10 and less than20 cigarettes per day), and 20 heavy smokers (those who smoked more than20 cigarettes per day). The duration of the smoking of the smokers was five years. Another 20 nonsmokers who never smoked & avoided passives smoking (those who avoided main stream smoke as well as side stream smoke) included in this study as control group. As such no dietary parameter was taken into account. A prior written consent of each subject was taken before including him in the study.

Sample size for each group was calculated on the basis of variation in HDL-C level among smokers and non-smokers with formula

$$N = \frac{(Z\alpha + Z\beta)^2 \ (\sigma_1^2 + \sigma_2^2)^2}{d^2}$$

Where, $\sigma 1 = 4.515$, $\sigma 2 = 3.215$ and $d = \max(\sigma 1, \sigma 2$

Type I Error $\sigma = 5\%$, Type II Error $\beta = 10\%$, Power Of Study= 90%, Sample Size N=20 for each group,and 20 for each Subgroup.

The following criteria has been used for exclusion:

1. Diabetes mellitus 2. Hypertension 3. Renal disease 4. Previous family history of CHD 5. Chronic hepatic dysfunction 6. Any endocrine disorder 7. Obesity 8. Ex- smoker 9. Alcoholics 10. Consumption of any lipid lowering drug /medicinal plant/ any other ayurvedic preparation.

Two groups were formed. Group 1 and Group 2. Group 1 comprised of control subjects and Group 2 comprised of smokers. Group 2 was further subdivided into 2A, 2B and 2C for mild, moderate and heavy smokers respectively.

Body weight and height of all subjects were measured and BMI was calculated as (weight/ height^2) (Kg/meter^2). 10 to 12 hours fasting blood sample of all the subjects was collected to measure serum HDL-C. HDL-C levels were measured by Phosphotungustic Acid method (13). Result was represented through mean ± SD. Statistical analysis was done by using SPSS version. P value < 0.05 was taken as statistically significant however P value > 0.05 was taken as statistically insignificant.

RESULTS

There were 80 subjects in the study. Of these 20 mild smokers, 20 moderate smokers and 20 heavy smokers in addition to 20 non-smokers. Their mean age ± SD was 36.3 ± 2.05, 37.1 ± 1.11, 36.25 ± 1.06, 36.70 ± 2.0 (Table 1). Body mass index (BMI) for the same was 21.19 ± 0.98, 21.94 ± 1.13, 21.86 ± 1.07, 22.03 ± 1.13 respectively. (Table 1). No significant difference was observed in age and BMI among control, mild smokers, moderate smokers, and heavy smokers (P value >0.05).

Character ↓	Control (Group 1)	Mild Smokers (Group 2A)	Moderate Smokers (Group 2B)	Heavy Smokers (Group 2C)
Number	20	20	20	20
Age (Mean ± SD)	36.3 ± 2.05	37.1 ± 1.11	36.25 ± 1.06	36.70 ± 2.01
BMI (Mean ± SD) kg/m^2	21.91 ± 0.98	21.94 ± 1.13	2186 ± 1.07	22.03 ± 1.13

Table 1: Showing Age, Body Mass Index (BMI) & Numbers in Control , Mild, Moderate & Heavy Smokers

For control the mean+ SD value of HDL-C was 49.96 ± 3.96 mg/dl. This value was found significantly different (P value<0.001) from mean+ SD value of HDL-C of mild smokers (37.32 ± 3.26), moderate smokers (33.40 ± 1.65) and heavy smokers (31.71 ± 2.48) (Table2& Fig 1).A significant difference was observed between the mean+ SD value of HDL-C of mild & moderate smokers & between the mean ± SD value of HDL-C of the moderate and & heavy smokers and between mean ± SD value of HDL-C of mild and heavy smokers (P value < 0.001) (Table 2 & Fig2).

Group	Mean Value of HDL-C ± SD (mg/dl)
Control (Group 1)	49.96 ± 3.96
Mild smokers (Group 2A)	37.32 ± 3.26
Moderate smokers (Group 2B)	33.40 ± 1.65
Heavy smokers (Group 2C)	31.71 ± 2.48

Table 2: Showing the Levels of HDL-C in Control, Mild, Moderate & Heavy Smokers

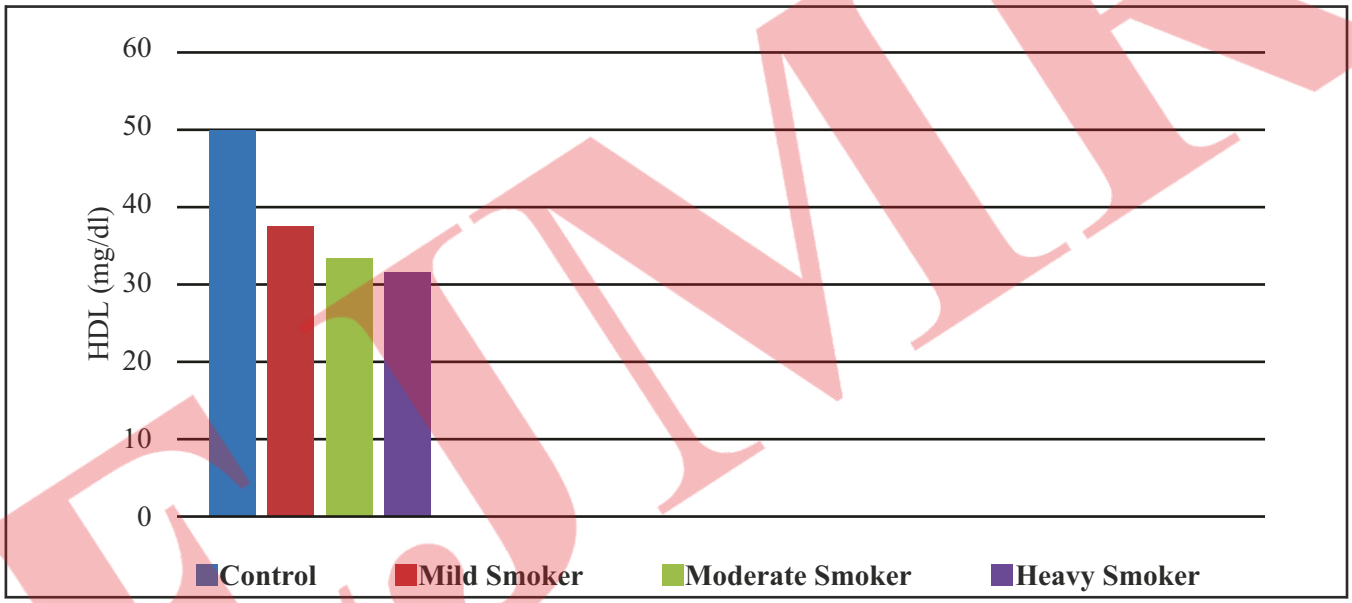

Fig 1: Comparison of Level of HDL-C in Controls & Smokers

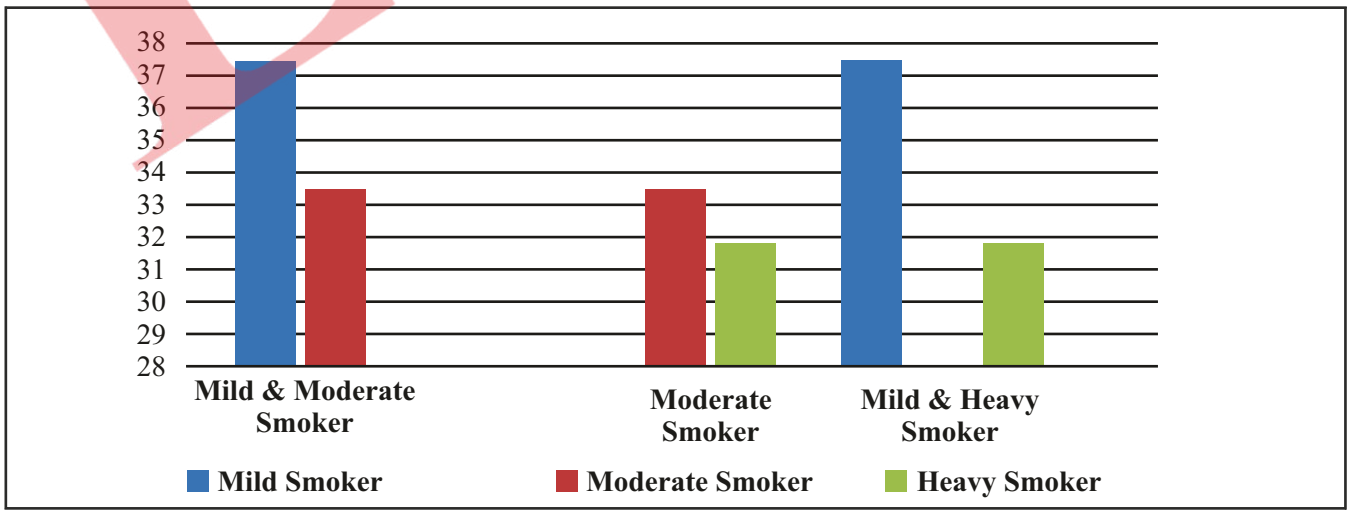

Fig 2: Comparison of Levels of HDL-C Between Group 2A & 2B, & 2C, 2A & 2C

DISCUSSION

It is well known that Cigarette smoke consists of a large number of chemicals and toxic substances which include hydrogen cyanide, tar, nicotine, carbon monoxide well as many irritants. Cigarette smoking is one of the leading cause of preventable morbidity that usually starts in adolescence and continues into adult life. In the developing world, tobacco consumption is rising especially among youth (14).

Smoking have a very important role in development of coronary artery disease besides this abnormal levels of serum lipid, lipoprotein and hypertension are also associated to the earliest stage of atherosclerotic coronary artery disease (15). It is well known that HDL –C is inversely correlated with the risk of atherosclerosis and coronary heart disease. Similarly various studies mentioned that smoking affects HDL-C by decreasing its level in serum, therefore this study was carried out to shade a light on which type of smoking (mild, moderate or heavy) decreases the HDL-C level.

Perusal of literature suggests that various studies have been done to show the relationship between cigarette smoking and its effects on lipid profile.. The harmfull effects of cigarette smoking are legion and reported prior. Various mechanisms responsible for alteration in lipid profile by smoking are (a) stimulation of sympathetic adrenal system by nicotine leads increased secretion of catecholamines that results increased lipolysis and elevated level of free fatty acids which results increased secretion of hepatic free fatty acids, triglycerides and very low density lipoprotein cholesterol in blood stream(6).(b) intake of food with high content of fat and cholesterol but lacking in fiber and cereal content(7) and (c) increased level of plasma homocysteine is responsible for oxidative modification of low density lipoprotein cholesterol and depletes high density lipoprotein cholesterol (8-9).

Furthermore, in the present study HDL-C was low in all type smokers as compared to control group and it is identical with most of the previous studies (16,17,18), On the contrary, Dirican et al . reported that there was no significant difference in lipid profile among smokers and non- smokers (19). However, other studies indicated that smoking is associated with the increased levels of total cholesterol, triglyceride, LDL-C, VLDL-C and decreased levels of HDL-C (20,21). Thus smoking may be responsible for atherogenic effect through blood lipid levels by decreasing the levels of HDL-C.

CONCLUSION

Smoking of tobacco is harmful not only to the person who smoke but also to the innocent bystander who unfortunately happens to share the same air as smoker.

Cigarette smoking causes depletion in the level of HDL-C in smokers. Increased intensity of smoking leads to decrease in anti atherogenic lipoprotein HDL-C. Smoking whether mild, moderate or heavy affects the levels of HDL-C by decreasing its concentration in serum and causes the risk of CHD. All the above finding support a need of educational programme about the risk of cigarette smoking. In addition to this the health policy makers should take an effective role in planning strategies to increase the awareness of the health risk of smoking and to illustrate the role and risk of smoking in the society.

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