

# A Comparative Study of Lipid Profile in Apparently Healthy Smokers and Non-Smokers.

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## ABSTRACT

**Background:** Smoking is considered as the single most important risk factor for coronary heart disease and cerebrovascular disease, mainly through its effect on accelerating atherosclerosis. It has also been observed that smoking causes increased serum cholesterol, triglyceride and beta lipoprotein levels and alpha lipoproteins. **Objective:** To compare the lipid profile among smokers and nonsmokers. **Method:** In this study the lipid profiles of apparently healthy non-smokers in the age group 20-55 was compared with that of smokers with relation to dose response and duration response relationship. **Result:** It was observed that the mean value of all lipids were significantly higher in smokers as compared to nonsmokers. Analysis of the data with regards to the duration of smoking also showed statistically significant increase in the mean levels of serum lipids with increase in the duration of smoking. There was also significant fall in the mean values of serum HDL in all groups of smokers in a dose response sequence when compared to that in non- smokers. However the fall was not clinically significant. Mean HDL in heavy smokers  $31.40 \pm 6.67$  as compared to  $48.47 \pm 6.88$  in nonsmokers. P value was  $<0.001$ . When the same characteristics were analyzed with regards to the duration of smoking again a statistically significant rise in LDL, VLDL cholesterol & a statistically significant fall in HDL cholesterol were observed when compared to that of non-smokers. **Conclusion:** The smokers are definitely exposed to the risk of high lipid levels and hence to the risk of cardiovascular diseases.

**Keywords:** Lipid profile, cholesterol, triglyceride.

## INTRODUCTION

Smoking is considered as the single most important risk factor for coronary heart disease and cerebrovascular disease, mainly through its effect on accelerating atherosclerosis. It has also been observed that smoking causes increased serum cholesterol, triglyceride and beta lipoprotein levels and alpha lipoproteins. The exact atherogenic mechanism of smoking is still not clear. However, in contrast to the above observations, some studies have concluded the contrary. There was no effect in modifying cholesterol or lipoprotein concentration during a 30-minute interval after smoking 20 filter tip cigarettes by habitual smokers or non smokers. No effect was observed even after heavy smoking for hours in the above group.<sup>[1]</sup> Brischetto et al<sup>[2]</sup> reported that there was no difference in lipid profile of persons smoking less than 15 cigarettes and non smokers. An editorial in American Heart Journal, 1980<sup>[3]</sup> stated that the relationship between smoking and ICH was unclear because the exact mechanism by which it causes atherosclerosis and increased risk of a heart attack was not established. However, Kannel of Framingham group had clearly emphasized the relationship between smoking and coronary artery disease.

A report in BMJ in 1987 stated that smoking lowered the levels of HDL cholesterol which was a negative risk factor for coronary artery disease, and increased the levels of Triglycerides in young smokers irrespective of age, sex, or obesity. It is postulated that there was a reduced intravascular lipolysis in smokers and increased exposure of the vascular endothelium to potentially atherogenic lipoproteins because of impaired clearance of triglycerides rich lipoprotein provided a mechanism by which cigarette smoking predisposes to cardiovascular disease. Many other studies including reports of Pozner H et al<sup>[4]</sup>, supported the view that serum cholesterol was significantly higher in healthy subjects who smoked as compared to non smokers.

## AIM OF THE STUDY

To compare the serum cholesterol, triglycerides, low density lipoprotein, very low density lipoprotein and high density lipoprotein levels in apparently healthy smokers and non-smokers with relation to intensity and duration of smoking.

## MATERIALS AND METHODS

The present study was carried out on 45 male smokers and 15 male non smokers between the age group of 20 and 65 years at the Mamata Medical College, Khammam. No patient had any evidence of diabetes mellitus, hypertension, and ischemic heart disease, renal or endocrine disease. So also,

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patients with a family history of hyperlipidemia were not included in the study. Subjects on lipid lowering agents or on diet restriction for any reason were also not included in the study. Ex-smokers and tobacco consumers were not taken up in the study. An ECG was done in all the subjects, as a baseline investigation for normal cardiac function. Duration of smoking was counted from the when the patients started smoking regularly. Venous blood samples were obtained after overnight fasting for blood lipids.

All the subjects were divided into 4 groups.

1. Non-smokers (n = 15), mean age 38 years, Subjects who have never smoked.

2. Mild smokers (n = 15), Mean age 42 years, Subjects smoking 1-10 cigarettes or bidis per day.
3. Moderate smokers (n = 15), Mean age 44 years, subjects smoking 11-20 cigarettes or bidis per day.
4. Heavy smokers (n = 15), Subjects smoking more than 20 cigarettes or bidis per day.

Each of the above group was further sub classified according to the duration of smoking as smoking for 1-10 years, and more than 10 years. All the patients were matched for B.M.I (Body Mass Index), age, weight. All the patients had almost similar food habits as regards to fat usage.

## RESULTS

**Table 1:** Lipid profile in the groups of smokers with relation to intensity smoking

Lipid Profile	Non smoker N=15	Mild smokers N=15	Moderate smokers N=15	Heavy smokers N=15
Total cholesterol	133.27±11.15	172.00 ±29.48 P<0.001	212.13± 23.13 P<0.001	229.00 ±64.44 P<0.001
Total triglyceride	87.60± 24.20	138.80± 38.25 P<0.001	155.93 ±50.72 P<0.001	186.00± 78.68 P<0.001
VLDLC	17.72± 5.50	24.44 ± 8.94 P<0.05	31.15 ±10.25 P<0.001	37.8 ±17.42 P<0.01
LDLC	89.36 ±16.37	112.57 ±25.19 P<0.01	135.62± 19.03 P<0.001	147.27 ±59.63 P<0.001
HDLC	48.47± 6.88	39.67± 8.93 P<0.01	37.53 ±8.93 P<0.01	31.40± 6.75 P<0.001

\* All values in mgm/100ml

NS = Insignificant, p < 0.05 = significant, p < 0.01 = highly significant, p < 0.001 = very highly significant.

**Table 2:** Lipid profile in the groups of smokers with relation to duration of smoking (1-10 years) compared to nonsmokers.

Lipid	Non-smoker	Mild smokers N = 6	Moderate smokers N = 6	Heavy smokers N=6
Total cholesterol	133.00±11.5	142.00± 14.82 P<0.001	154.12 ±15.12 P<0.001	194.00 ±21.48 P<0.001
Total triglyceride	87.60± 24.20	134.80 ±32.25 P<0.001	140.00 ±40.62 P<0.001	154.00± 50.68 P<0.001
VLDLC	17.72 ±5.50	20.22 ±6.50 P<0.01	24.15± 8.14 P<0.001	28.16 ±14.42 P<0.01
LDLC	89.36± 16.37	100.44± 20.19 P<0.01	120.61± 12.03 P<0.001	127.50 ±17.81 P<0.001
HDLC	48.47± 6.88	42.47 ±6.93 P<0.01	38.43± 8.33 P<0.001	36.60± 6.48 P<0.001

\* All values in mgm/100ml

NS = Insignificant, p < 0.05 = significant, p < 0.01 = highly significant, p < 0.001 = very highly significant.

**Table 3:** Lipid profile in the groups of smokers with relation to duration of smoking (>10 years) compared to non-smokers.

Lipid	Non-smoker	Mild smokers N=6	Moderate smokers N=6	Heavy smokers N=6
Total cholesterol	133.00± 11.15	152.00 ±16.82 P<0.001	171.00 ±38.12 P<0.001	206.00 ±60.00 P<0.001
Total triglyceride	87.60 ±24.20	144.60 ±36.25 P<0.001	150.00 ±45.80 P<0.001	160.00 ±62.80 P<0.001
VLDLC	17.72 ±5.50	24.21± 7.40 P<0.05	28.15± 9.12 P<0.01	30.90 ±12.62 P<0.001
LDLC	89.36 ±16.37	110.11± 22.11 P<0.01	131.00± 11.11 P<0.01	140.84± 56.50 P<0.01
HDLC	48.47± 6.88	40.67± 7.20	38.10 ±8.11	35.30± 6.22

\*All values in mgm/100ml

NS = Insignificant, p < 0.05 = significant, p < 0.01 = highly significant, p < 0.001 = very highly significant.

In this study lipid profiles of apparently healthy non-smokers in the age group 20-55 was compared with that of

- 15 smokers smoking 1-10 cigarettes or biddies/day
- 15 smokers smoking 10-20 cigarettes or biddies /day
- 15 smokers smoking >20 cigarettes or biddies /day

The mean values of total cholesterol, total triglycerides, HDL cholesterol, VLDL cholesterol & LDL cholesterol are given in Table 1.

The lipid profiles of non smokers were also compared with smokers smoking for

- 1 -10 years (mean values in [Table-2])
- >10 (mean values in [Table 3])

It can be observed that the mean value of serum cholesterol was significantly higher in smokers as compared to non – smokers [Table 1]. The cholesterol levels were higher in all groups of smokers (mild, Moderate & heavy), but they were significantly (clinically & statistically) elevated in heavy smokers. Mean value of Total cholesterol among smokers was 229± 64.44 as compared to 133± 11.15 in non smokers (p < 0.001). Analysis of the data with regards to the duration of smoking also showed statistically significant increase in the mean levels of serum cholesterol with increase in the duration of smoking [Tables 2 & 3]. The P Value was < 0.001. There was also a clinical & statistically significant increase in serum triglyceride levels in smokers when compared to non-smokers especially so in heavy smokers. Mean value of serum triglyceride was 186± 78.68 among smokers as compared to 87.60± 24.20 in non smokers (p < 0.01). A significant rise was also noted in the triglyceride levels of smokers smoking for a longer duration. (Mean 160± 62.80) as compared to that of non smokers (Mean 87.60± 24.20) (p < 0.01). The mean values of serum LDL & VLDL cholesterol were found to be raised

significantly in all groups of smokers when compared to non- smokers. (LDL = 147.27± 59.63 as compared to 89.36±16.37, p<0.01). (VLDL = 37.87±17.42 as compared to 17.72± 5.50 p<0.001). There was also significant fall in the mean values of serum HDL in all groups of smokers in a dose response sequence when compared to that in non-smokers. However the fall was not clinically significant. Mean HDL in heavy smokers was 31.40±6.67 as compared to 48.47±6.88 in non smokers (p < 0.001). When the same characteristics were analyzed with regards to the duration of smoking again a statistically significant rise in LDL, VLDL cholesterol & a statistically significant fall in HDL cholesterol were observed when compared to that of non-smokers.

## DISCUSSION

Smoking in different form, was found to be a strong risk factor for various clinical entities, especially for cardiovascular morbidity and mortality. This excess morbidity is thought to be due to accelerated atherosclerotic coronary artery disease. The exact mechanism by which smoking induces atherosclerosis is not clear. Various schools of thought have been postulated. The most significant among them seems to be the conclusion from various studies that smoking induced dyslipidemia causing elevated S. cholesterol, S. triglycerides, LDL, VLDL cholesterol & decreased anti atherogenic HDL cholesterol and accelerating atherosclerosis. Several controversies have arisen on this conclusion. The controversy started with the causal hypothesis of the surgeon general's report on smoking and health in 1980. Apart from statistical association of smokers with myocardial infarction, sudden death and atherosclerosis, the causal connection was not clear. It had been stated that relatively little is known about the mechanism

by which smoking enhances atherogenesis or increases the risk of heart attack. Since then various studies have been done and various mechanisms have been put forward. The present day study shows a significant elevation in the mean cholesterol levels in a dose response fashion. This is in accordance with Pozner H, et al<sup>[4]</sup> who demonstrated significantly elevated serum cholesterol in healthy smokers. Karvonen et al<sup>[5]</sup> noted a direct correlation between the cholesterol and smoking. This finding is also in agreement with Rastogi et al<sup>[6]</sup> and Muscat et al.<sup>[3]</sup> Muscat et al<sup>[3]</sup> have demonstrated a dose response risk in the S. Cholesterol when analyzed in mild, moderate and heavy smoker However, Brischetto et al<sup>[4]</sup> did not find an elevated S. Cholesterol in their studies. The present study also found significantly elevated serum triglycerides in all groups of smokers. The values are statistically as well as clinically significant, especially in heavy smokers. Pozner et al<sup>[4]</sup> also demonstrated elevated S. triglycerides in smokers. This finding is also in accordance with Rastogi et al<sup>[6]</sup>, who demonstrated significantly elevated S. triglycerides in a dose response sequence in mild and moderate smokers. However, Brischetto et al<sup>[2]</sup> did not find elevated S. triglycerides in smokers smoking less than 15 cigarettes/day. In our study the mean LDL, VLDL levels are in accordance with the findings of Rastogi et al<sup>[6]</sup>, A.K.Sinha et al<sup>[7]</sup> and RK Tilwani et al<sup>[8]</sup> in a study done in north India. The present study showed declining HDL cholesterol in mild, moderate and heavy smokers when compared to non- smokers. The decline was observed to be statistically significant and occurred in a dose response sequence. Rastogi et al<sup>[6]</sup>, A.K.Sinha et al<sup>[7]</sup>, R.K.Tiwani et al<sup>[8]</sup> had observed similar findings in their studies. Whitehead TP and Robinson D<sup>[9]</sup> demonstrated a significant negating effect of smoking on HDL cholesterol in their studies in mild and moderate drinkers. They showed that smoking negated the observed increase in HDL cholesterol in mild and moderate drinkers.

## CONCLUSION

The smokers who are definitely exposed have the risk of high lipid levels and hence the risk of occurrence of cardiovascular diseases are higher with smokers.

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**How to cite this article:** Rampure DM, Anudeep K, Appa R, Krishna MK. A Comparative Study of Lipid Profile in Apparently Healthy Smokers and Non-Smokers. Ann. Int. Med. Den. Res. 2016;2(1):121-24.

**Source of Support:** Nil, **Conflict of Interest:** None declared